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*Kuhn, H.S.: Tr. Am. Acad. Ophth., p. 432, (March-April) 1951.

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Frontispiece illustrating article by Brendan D. Leahey

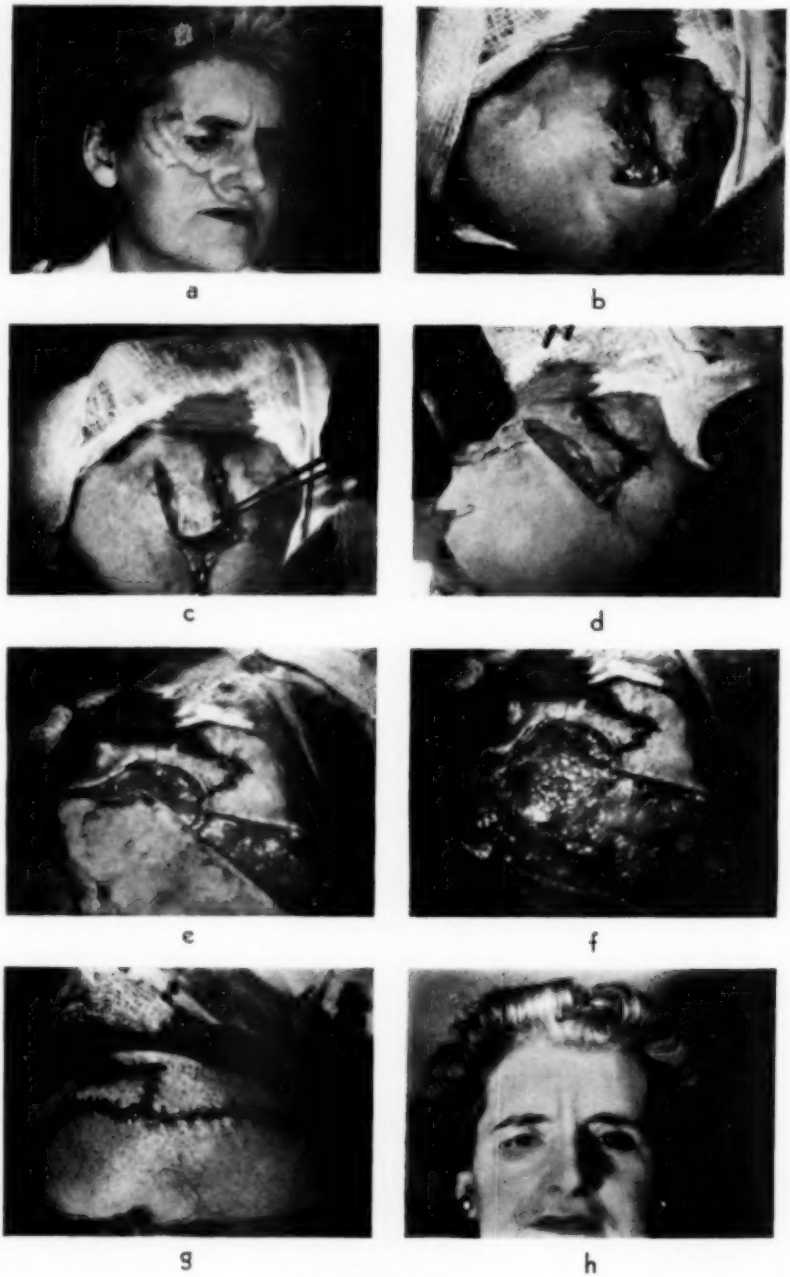

FIG. 19 (LEAHEY).
(NOTE THAT ILLUSTRATIONS (A) AND (H) HAVE BEEN REVERSED FROM LEFT TO RIGHT.)

AMERICAN JOURNAL OF OPHTHALMOLOGY

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*Symposium: Ocular Injuries**

* * *

THERMAL BURNS OF THE EYE AND ADNEXA

BRENDAN D. LEAHEY, M.D.
Lowell, Massachusetts

Thermal burns of the eyes or upper face are acquired in a multitude of ways. The commonest cause in children is probably boiling water or coffee. Accidental ignition of clothing and fires in homes and institutions have resulted in facial burns to large numbers of people. In industrial plants, hot solutions of many kinds and molten metals take their toll. Particularly severe burns occur in epileptics falling into open fireplaces or against hot stoves or radiators. Deep burns of the exposed areas such as the face also result frequently from explosion of gasoline in automobile and airplane accidents.

In modern warfare the ignition of oil and gasoline aboard ships, the use of highly volatile gasoline for all purposes, the incendiary bombs and flame throwers tend to increase the number of thermal burns. Davis[1] found that burns constituted 60 percent of all Pearl Harbor casualties. Even in a typical peace time year, the incidence of burns among eye injuries of Navy personnel was about 20 percent according to Trexler.[2]

Burns of the face alone seldom endanger life, but they are potentially more serious than considerably larger burns elsewhere. Besides endangering sight, even small facial burns may cause more visible distortion than tremendous burns on other parts of the body.

CLASSIFICATION OF BURNS

In this country it is customary to classify burns in three categories according to depth:

1. *First degree,* simple erythema.
2. *Second degree,* partial destruction of the skin without destruction of all epithelial cells (characterized by bleb formation).
3. *Third degree,* destruction of the full thickness of the skin.

Superficial second-degree burns may heal without scarring in about two weeks. Deeper second-degree ones result in slough, cleavage, fibrous tissue proliferation, and scarring. In the third-degree wounds, epithelization can start from the periphery or grafts only after the slough has been eliminated. Spontaneous elimination requires three to five weeks.

It is frequently impossible to estimate the

* Presented at the clinical session of the American College of Surgeons, San Francisco, November 5, 1951.

←

FIG. 19 (LEAHEY). SURGICAL TREATMENT OF A THIRD-DEGREE BURN OF FACE AND EYELIDS.

A. BADLY BURNED. DENUDED AREAS HAD BEEN COVERED WITH SPLIT-THICKNESS GRAFT THREE WEEKS AFTER BURN. MASSIVE SCARRING AND ECTROPION SHOWN THREE MONTHS LATER.

B. LID-MARGIN SPLIT. LID SKIN SEPARATED FROM SCAR. TARSUS SHORTENED BY A "V" RESECTION AND TEMPORAL SKIN TRIANGLE REMOVED AS IN KUHNT-SZYMANOWSKI PROCEDURE.

C. LID SKIN FREED FROM SCAR TISSUE INFERIORLY.

D. LID HAS BEEN SUTURED INTO PLACE. KELOID SCAR EXCISED FROM UPPER LIP UP TO THE TRANSVERSE SCAR.

E SCAR TISSUE EXCISED LATERALLY TO REGION ABOVE EAR.

F. SKIN OVER ENTIRE LOWER FACE UNDERMINED TO MAKE FREELY MOVABLE FLAP.

G. END OF OPERATION. VERY FINE SUTURE LINES REPLACE ORIGINAL WIDE UNSIGHTLY SCARS.

H. SIX MONTHS LATER. SCARS ALMOST IMPERCEPTIBLE. EXCELLENT FACIAL MOBILITY.

depth of a burn accurately until at least a week or two has passed. Upon first observing the early lesion, we may say that a burn area which is dry, hard, dead-white, firm or leathery to the touch, or charred, is probably third degree. The presence of blebs or bullae, oozing of plasma, and visible corium, usually indicate a second-degree burn.

From the standpoint of general prognosis the total area of the burn is more important than the depth of the burn. Up to 1941 it was conceded that a burn involving more than 50 percent of the body surface would prove fatal. Now a patient with a burn of more than 70 percent, even if the greater proportion is full thickness, is not necessarily doomed.

Systemic effects

It is outside the province of this paper to discuss the general effects of severe burns. Passing mention, however, should be made of certain salient points:

1. Primary shock which may be present immediately after injury is similar to any surgical shock.

2. Secondary shock (burn shock) may begin almost immediately after the burn or may be delayed for several hours or days.

Shock can be expected in burns which involve more than 15 percent of the surface area of healthly adults and more than 10 of the surface of children or aged persons. It is due to the large loss of plasma into the tissues in and surrounding the burned area and to a small loss of plasma to the exterior and in other parts of the body.

The signs of secondary shock are pallor, sweating, thirst, shallow respiration, vomiting, restlessness, and occasionally convulsions. Blood pressure drops, and laboratory tests show hemoconcentration. Fluid replacement by administration of plasma, serum, saline, or whole blood is necessary.

3. Occasionally burn patients may die in the absence of marked fluid loss and its associated changes. The collapse of patients with small burns or those with large injuries after the time when shock would still be active is explained by many authors on the basis of acute toxemia. During this third stage, necrosis has been noted in the brain, liver, heart, and adrenals.

4. Significant temperature elevation after the third or fourth day almost always means infection at the site of the burn. This may signal the onset of septic toxemia.

Local treatment

Burns of the upper face present certain special problems not met elsewhere. Chief of these is the marked tendency for early contraction with deformities of the lids and mouth, and the need for protecting or possibly treating the eye simultaneously with the facial injury.

When the patient is first seen, the burned area should be protected with a sterile dressing. If the patient is conscious and in pain, morphine or codein may be used. If shock is a factor, morphine is given intravenously to insure immediate absorption. The burned area is diagrammed and an attempt is made to estimate as far as possible the degree and depth of the injury.

If shock is present or if shock is anticipated from the extent of the burn, intravenous therapy is started.

After shock treatment has been instituted, the regular ointment dressings are applied. If the wound is extremely dirty, it may be gently cleaned with white soap or a detergent but vigorous cleansing or debridement are contraindicated.

Formerly the outer layers of the blebs were removed as it was thought that the fluid of the unpunctured blebs would become purulent. Actually, however, the roof of the bleb sometimes serves as an additional barrier against wound infection.

"It is probable also that the blister fluid has a relatively high oxygen content and that infection of unruptured blebs produced by the normal bacterial habitants of the skin or an occasionally virile organism can be controlled by antibiotics. It has been possible, for instance, to prove free permeation of the

sulfanilamides into the wound of these blebs by analyzing the fluid periodically. Healing beneath either ruptured or unruptured blebs has been found to occur with equal rapidity."[3]

Since World War II, the most popular local burn dressing has been fine mesh gauze impregnated with petrolatum. Boric-acid ointment is also popular, but, on more extensive surfaces, may result in toxic absorption. The ointment dressings are applied to the burn area and over this initial layer a bulky layer of fluff gauze is followed by a firm wide bandage, where possible, to give gentle compression. The pressure dressing minimizes edema and cuts down the incidence of infection. On extensive facial burns the entire head may be enveloped with the exception of the nose and mouth. Another method of applying light pressure on the face is a stockinette over the head with openings for nose, mouth, and undamaged eye.

If the eyeball has not been damaged, ointment may be instilled in the eye and the eye left covered for a week or more until the dressing is to be changed.

The edema which accompanies facial burns often reaches tremendous proportions. Eyelids may be extremely puffed out and the lips and cheeks have a balloonlike appearance.

Dressings may remain undisturbed as long as they are dry and clean and temperature is normal. On deep second- and third-degree burns, however, dressings are likely to become saturated with exudate somewhere between the fifth and 10th day and, if the burn is extensive, the patient's temperature rises. This usually means infection.

In general, we plan to remove the dressing between the seventh and 10th day and inspect the wound. At this time the more superficial second-degree burns are healed. On deep second-degree burns, the thin slough which has formed may be carefully removed.

"Pearly white, growing epithelium, or small granulated areas alternating with islands of spreading epithelium are seen beneath the slough. Third-degree burns appear as dark yellow, brown, or black slough separated from the surrounding tissue."[4]

In case of infection the dressings are changed daily, or even more often. Antibiotics are used routinely beginning the first day on all severe burns.

Skin grafting

With full thickness burns and deep second-degree burns with sloughing, convalescence is speeded up and contractures and late deformities are minimized by skin grafting as soon as possible. If the sloughs are allowed to separate spontaneously, the area may be ready for grafting in about three weeks, but often not till much later.

On other parts of the body continuous dressings of Dakin's solution help to get rid of the infected slough, but these are not practical close to the eye. Application of pyruvic-acid paste[5] is one good method of greatly accelerating separation of this slough. On occasional small burns, surgical excision of the slough is advantageous.

When the slough separates exposing the underlying granulation, the area is ready for grafting. If the wound is grossly infected and covered with exuberant granulations, all crushed and necrotic tissue is removed and the area covered with fine sterile gauze. Saline dressings are then applied every four hours over this gauze for several days.

After about a week, the fine mesh gauze is removed, all exuberant granulations are cut down to a firm base, and the gauze and wet saline dressings are resumed. Granulations are never scraped off but are sliced off in sheets leaving a small yellow base. After about two more days of these dressings, even the unfavorable wounds are usually ready for grafting.

Split-thickness graft

On these granulating surfaces on the face, the split-thickness graft is the type usually advisable. Reverdin or pinch grafts take easily, but leave a very irregular unsightly scar. Full-thickness grafts (either free or

pedicle) do not take well on granulation tissue.

The split-thickness graft gives a fairly good cosmetic result on the face, but it leaves a thinner, less flexible covering than the full-thickness graft, and its general appearance, texture, and mobility are definitely inferior to that of the full-thickness ones. It has the advantage over the full-thickness ones in that it "takes" in an extremely high percentage of cases, and does especially well over clean granulation tissue. It will even take on the periosteum of the malar bone or orbital rim. It is also easily available and is less apt to be affected by minor infection. Thus, while successful full-thickness grafts usually give a better-looking result around the cheeks and eyelids it is usually safer to employ the split-thickness one for grafting at this intermediate stage.

On small burns on the lids and cheek, however, it is often desirable to excise clean granulation tissue, obtain a sharp margin of good skin, and employ a full-thickness, free, or rotating graft rather than wait for a probable late secondary repair.

A thick split graft can be taken with a dermatome in any size desired up to four by seven inches. The thinner the split graft the more likely it is to take successfully. The thicker ones, however, have more normal texture and are also less apt to show marked contracture.

When the granulation tissue is young and firm, it is not disturbed; but when the recipient area is covered with old exuberant granulation tissue, the granulations are shaved off down to a firm yellow base. Bleeding is controlled by warm saline packs and the graft is then applied. If there is any bluish epithelium around the edges of the defect, it is removed; otherwise it is disfiguring later.

The graft is fixed in place with fine silk sutures. We have been using dry gauze dressings with general pressure, leaving the dressing in place for seven days. Vaseline gauze used elsewhere is apparently equally effective. At the time of the first dressing, any area of skin not taking well will be moist and blistered and look either darker or quite white.

Immediate full-thickness grafts

Practically all deep burns that go on to slough have some degree of infection. Since the ordinary routine of treatment and skin grafting just described is a prolonged one, immediate grafting is preferable on certain cases. If an area of skin close to the eye is a probable third-degree burn, and if the patient is in good general condition, immediate excision of this area and replacement with a graft will often give quick restoration of function with minimal deep contracture and deformity. If the wound is clean and not too large, a full-thickness graft is preferred.

"The best base on which to lay grafts is the one left after excision during the first hours after injury. This base may be made up of fatty or areolar tissue, fascia, muscle, or periosteum. Even if the base is grossly edematous and exudating water, grafts may be laid directly on it with assurance. The graft will not take, however, if a thin layer of dead tissue is left; that is, if the excision is not deep enough or the base is inflammatory. The longer the delay before excision the greater will be the inflammation."[3]

During excision of the burn wound, control of hemorrhage is sometimes annoying. If done the first day, during the period of development of edema, the bleeding is localized and easily controlled. After several days, an inflammatory reaction develops beneath the burn slough. Where the line of excision is carried into this inflammation, bleeding may be profuse and extremely troublesome.

When a graft of any type is applied in the area of the lids and especially below the lower lids, it is advisable to suture the lids temporarily with three mattress sutures tied over tiny rubber plates (fig. 1), or the lid margins may be scraped and sutured directly with 5-0 intramarginal silk mattress sutures (fig. 2). This serves a triple purpose of helping immobilize the lids, reducing contracture

of the grafts, and protecting the cornea. On these burns in the area of the eyes, of course, the result of burn contracture is usually extreme cicatricial entropion. Pesides the cosmetic defect, this may also result in marked visual loss due to corneal exposure.

When the burns affect the conjunctiva and cornea rather than the surrounding skin and tissue, a completely different set of problems arise. Fortunately, on most burn patients, the wink reflex is quicker than the flash or flame so that only a small percentage of facial burns actually do have serious burns of the eye itself (fig. 3).

Shenk, Silcox, and Godfrey,[6] for instance, reported that, of 451 eye patients treated on a hospital ship, 212 had burns of the eyelids, but only 27, or 12 percent, had burns of the cornea.

In general, my experience has been that burns from actual flames, as in a burning building or gasoline fire, seldom cause more than mild erythema to the eyeball. Spatter burns from boiling liquids and injuries from molten metals, however, will frequently involve the cornea and conjunctiva. Electrical flash burns also will usually leave a first- or second-degree burn of the cornea and conjunctiva the exact shape of the palpebral aperture (fig. 4).

With mild superficial burns of the conjunctiva and anterior cornea, relatively simple treatment usually suffices. Mild antiseptic eye ointment such as White's ointment (1:3,000 mercury bichloride) is applied several times a day and atropine is used if the eye is markedly inflamed. The eye does not need to be covered unless the patient finds it more comfortable, or a dressing is necessary also for a lid burn. Healing is usually quicker than with chemical burns and there is less tendency for chronic inflammation and late vascularization.

Full-thickness burns of the cornea and

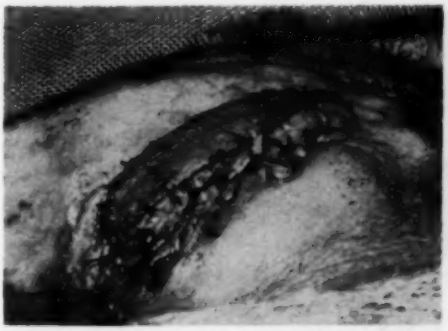

Fig. 1 (Leahey). Full-thickness graft of the upper lid. Lids are sutured with three mattress sutures tied over tiny rubber plates.

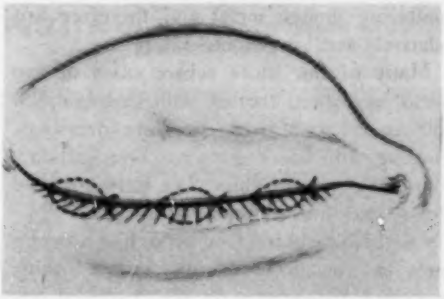

Fig. 2 (Leahey). Direct sutures with intramarginal silk mattress sutures.

Fig. 3 (Leahey). A second-degree burn of the entire face from gasoline. The burn was superficial and healed without visible scarring.

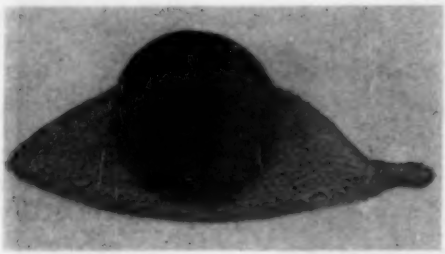

Fig. 4 (Leahey). Electrical flash burns are usually the exact shape of the palpebral aperture.

sclera are infrequent, though occurring more often on lid margins. In our experience the full-thickness burns have usually been from spattering molten metal and therefore are relatively small in extent.

Many of our more severe cases of eye burns have been treated with hydrosulphosol* in oil and gentle pressure dressings. Healing with this regime has been satisfactory but not remarkable. We have not used this preparation on burns of the skin.

On these cases we frequently find a severe burn in the lower fornix with a white, procelain appearance on the inside of the lower lid and the adjacent sclera and conjunctiva.

This porcelain appearance indicates that the circulation has been completely destroyed. If these areas are on the sclera or cornea, they are likely to perforate one to four weeks later. It is probable also, when a large area of the limbus is coagulated, that corneal nutrition and healing power are seriously impaired.

Cases of delayed descemetocele and threatened perforation of the cornea are more common after chemical than thermal burns. If, as in a case treated recently, the conjunctiva has healed before corneal perforation is imminent, suturing of the eyelids for several weeks will support the cornea and may allow it to cicatrize over without rupture, and without formation of symblepharon.

* Hydrosulphosol is the registered trademark of E. C. Lientz and Co., Inc., for their brand of calsulfhydryl.

Symblepharon formation

When there is involvement of two opposing surfaces, symblepharon will almost certainly quickly form if preventive steps are not taken. A severe burn of one surface, however, will seldom present this complication as long as the opposing surface is undamaged. In some cases symblepharons can be so complete as to fuse the entire lid surfaces of both eyes to the surface of the eyeball. Prevention of these symblepharon, therefore, is a major point in the treatment of conjunctival burns.

When the burns are of limited extent, it is usually sufficient to have the patient exercise the eyeball in all directions several times daily. If the burn extends down into the lower fornix, as it usually does, or into the upper fornix, the growth of symblepharon may be minimized by daily passage of a blunt pointed glass rod along the fornix each day after instillation of pontocaine. When there is extensive involvement of both the eyeball and lid surfaces, more radical treatment is needed.

Deep burns of globe

Prevention of symblepharon by use of plastic conformers is occasionally mentioned in the literature, but these are very unsatisfactory. They are not tolerated well by the patient and they may cause serious damage to the cornea. The best method on the severe burns is an immediate mucous-membrane graft. This may be conjunctiva from the other eye (figs. 5 and 6); or buccal mucous membrane may be used.

A small piece of buccal mucosa (up to 1.0 by 5.0 cm.) can be taken from the inner surface of the lower lip (fig. 7) and larger pieces up to size 1.5 by 5.0 cm. can be obtained from each side of the mouth (fig. 8).

The graft is stretched on a gloved finger, trimmed of submucous tissue with scissors, and is then applied directly to the denuded area and sutured with 6-0 black silk (fig. 9). If the entire bulbar conjunctiva is burned, two horseshoe-shaped grafts can be used to

encircle the cornea (fig. 10). A drop of atropine is instilled in the eye, White's ointment is applied, and a double eyepad.

Buccal mucous membrane, while functionally satisfactory, does not give a pleasing appearance in the eye. It remains permanently thickened and slightly red in color; and should be reserved therefore for the more desperate cases and ones in which conjunctiva is not available. The buccal wounds usually can be closed easily with practically no attempt at undermining. Even when left unsutured they usually heal spontaneously in a short time.

When there is no danger of perforation of the eyeball and the burns involve the lower fornix area, another method of symblepharon prevention is as follows:

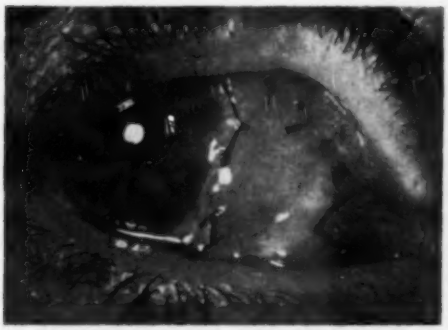

Fig. 5 (Leahey). A free conjunctival graft nine days after insertion.

Fig. 6 (Leahey). Opposite eye, showing donor site of conjunctival graft illustrated in Figure 5.

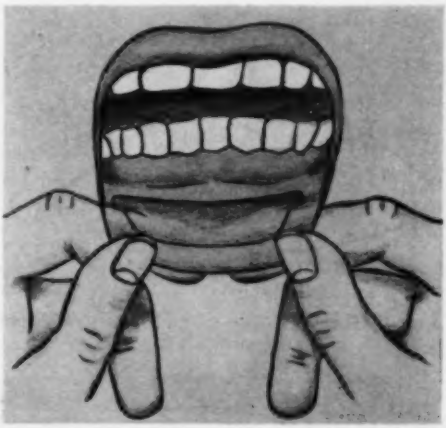

Fig. 7 (Leahey). Donor site for mucous-membrane graft from lower lip.

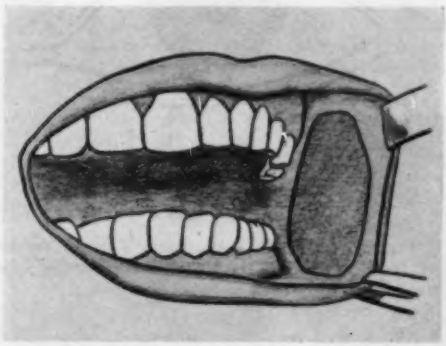

Fig. 8 (Leahey). Donor site for mucous-membrane graft from cheek.

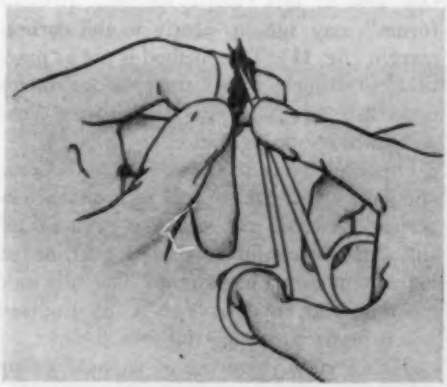

Fig. 9 (Leahey). Method of trimming submucous tissue from buccal mucosa.

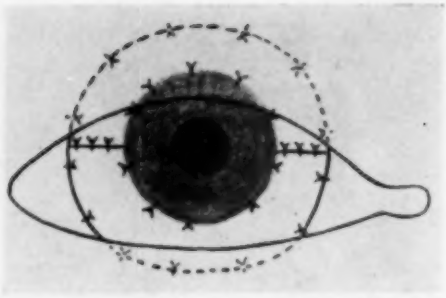

Fig. 10 (Leahey). Use of two horseshoe-shaped grafts to encircle the cornea.

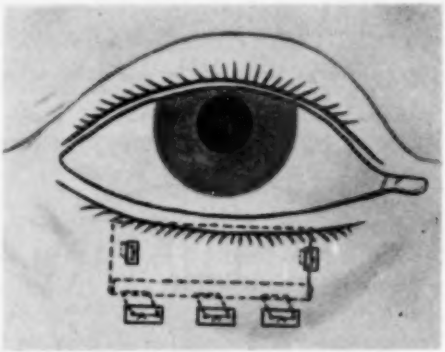

Fig. 11 (Leahey). A temporary artificial fornix to reduce symblepharon formation. This may be used in lower but not in upper fornix.

A piece of very thin rubber may be sutured in the lower fornix by two mattress sutures running out through buttons in the cheek. One side of this folded, rubber, "artificial fornix" may run up nearly to the corneal margin (fig. 11). This method is not as practical in the upper fornix since the eye rotates upward in sleep and the friction may cause additional corneal irritation.

Other methods reported for these cases, but with which I have had no personal experience, include use of rabbit peritoneum[7] and cadaver conjunctiva[8] and grafting of human amniotic membrane. The amniotic membrane as used by Sorsby[9] in England was a preprepared dry fat-free product.

Tissue culture studies in wound healing with amniotic membrane show that the tissue cells continue to grow, being neither stimulated nor inhibited by the presence of amniotic membrane. It would seem, therefore, that a graft of amniotic membrane over a raw surface, while not preventing growth of the proliferating edge of the wound, would guide such proliferation toward the wound surface. This work indicates that it might be a logical thing to use on these cases, but at the present time it is not readily available in the proper form in most American medical centers.

Sorsby treated 58 cases of conjunctival burns with amniotic membrane grafts (55 chemical burns and three thermal burns) with excellent results. He found it necessary, however, not to cover the corneal surface as these grafts affected the cornea adversely. He reported that, when the bandages are removed after 48 hours, the graft is no longer visible. "It has become transparent and forms a layer over the conjunctival surface."

In recent months our treatment of conjunctival and corneal burns has included the local use of ophthalmic cortisone solution (0.5 percent) at each dressing, and four times daily when dressings have been removed. On corneal wounds, especially, cortisone cuts down irritability of the eye and consequently the tendency for the leukoma to vascularize. After extensive burns of the skin, cortisone or ACTH administered systemically during the first few days appears to reduce the severity of the inflammatory reaction, and thus lessen the tendency for burn shock. They are apparently of little value, however, in later treatment of skin burns.

Late repair

Unfortunately, considerable treatment may be necessary on many burn cases even after the primary wound has finally healed. The three main problems are: (1) Treatment of vascularized leukoma of the cornea; (2) removal of symblepharon; (3) treatment of cicatricial ectropion and other cosmetic defects of the lid and face.

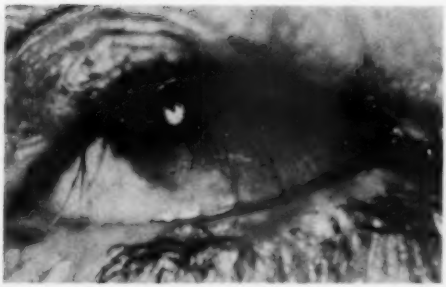

Fig. 12 (Leahey). Old gasoline burns of cornea and eyelids resulting from airplane crash.

Vascularized leukoma

Corneal burns going through Bowman's membrane leave permanent corneal scarring. Small central ones usually heal without vascularization. Large burns, however, or small burns near the limbus, invariably have progressive vascularization of the scar for many months (fig. 12).

If the center of the cornea is not involved, no treatment is necessary on these vessels since, unlike those of rosacea and trachoma, the vessels usually show no tendency to spread beyond the affected area. If corneal graft is to be considered later, however, the vascularization should be eliminated by beta irradiation after the cornea has epithelized over.

After the first beta treatment it is also desirable to coagulate with an electrical needle any large trunks running in from the limbus, as it is difficult to close these larger vessels with ordinary doses of beta irradiation. Since the beta works best on young vessels, it is most effective when used one to two months after the original burn.

When vascularization of the scar is very extensive, the conjunctiva should be resected for four mm. back of the limbus and the sclera scraped clean of superficial vessels.

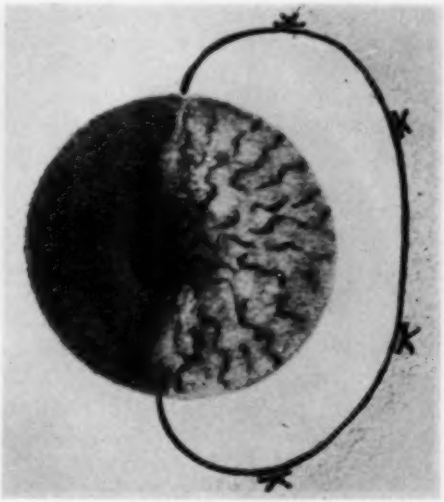

Fig. 13 (Leahey). Elimination of vascularized pannus on cornea. The cornea is stripped, conjunctiva is resected for four mm. around the limbus and beta irradiation is started three days later.

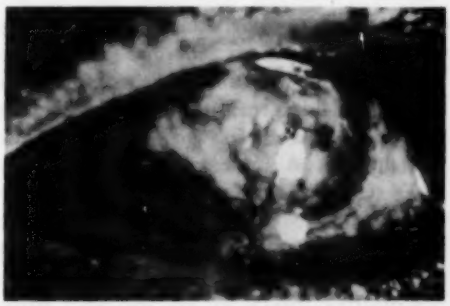

Fig. 14 (Leahey). Very thick vascularized scar of entire cornea from burn 40 years earlier.

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Fig. 15 (Leahey). Closeup view of cornea shown in Figure 14 after complete stripping of cornea, excision of conjunctiva all around the limbus, and beta irradiation.

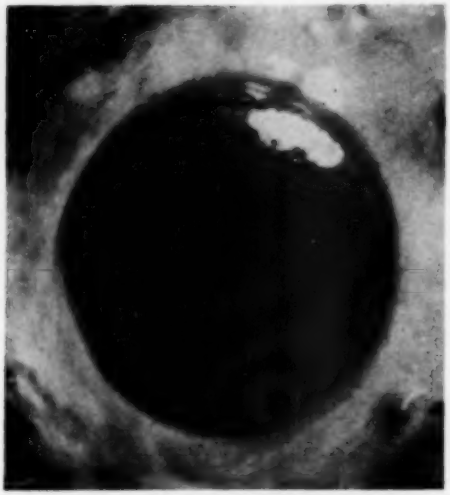

Fig. 16 (Leahey). Same case as shown in Figures 14 and 15, after successful corneal graft. The white areas are tags of Descemet's membrane left when opaque cornea was removed.

This area is left bare, and the conjunctiva is sutured to the sclera four mm. back of the limbus. Beta irradiation is started three days later over this area and the cornea before the new vessels have a chance to grow back in (figs. 13, 14, 15, and 16).

Symblepharon removal

There are many methods of removing symblepharons. As mentioned previously, conjunctiva makes by far the most satisfactory graft. Since buccal mucosa retains its rosy color, it is wise to use conjunctiva as far as possible at least in the area of the palpebral aperture. Clay and Baird[10] used grafts from the prepuce and labia minora for the correction of symblepharon and after removal of large growths from the conjunctiva. These grafts are conspicuous, have an unpleasant odor, and desquamate.

Late repair of the lids and face

This is a big subject upon which we can barely touch in this presentation. Whether the original healing takes place by means of spontaneous epithelization or coverage with a split-thickness graft, a certain amount of contraction takes place. This contraction is due more to contracture of the granulomatous base upon which the graft is placed than to actual contraction of the graft itself. Many burns also have prominent, shiny, keloid-type scars where grafts have not been applied. When extensive scarring is near the lid, grotesque, extreme ectropion may result.

The first principle in the repair of these deformities is to excise all of this scar tissue through its full depth. The second principle is to close the defects thus made in the tissue with full-thickness free grafts or with full-thickness sliding or rotating grafts rather than falling back on the split-thickness graft so widely used for repair during the intermediate granulating stage. The split-thickness graft has a more stiff and shiny appearance than the full-thickness graft and it will not match the surrounding skin in texture or color as well as a full-thickness one.

When full-thickness free grafts are utilized, small ones should always be taken from the upper lid skin when possible (fig. 17). Larger ones may be taken from just above the clavicle (fig. 18); and more extensive ones from one side of the abdomen. In general, matching of these grafts with the facial skin is in that order. Skin may also be used from behind the ear, but this is frequently more pink than ordinary lid skin.

Since ectropion is the most common late

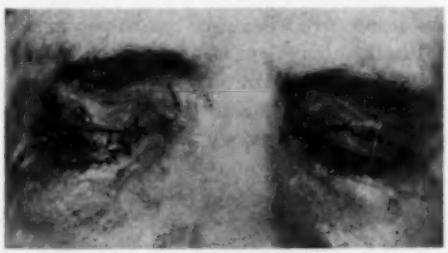

Fig. 17 (Leahey). A full-thickness graft taken from the opposite upper lid for cicatricial ectropion.

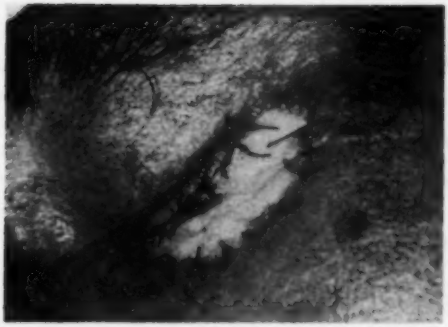

Fig. 18 (Leahey). A full-thickness graft of the lower lid taken from just above the clavicle.

deformity, it follows that most of the routine surgery is designed to cure this defect. The ordinary Kuhnt-Szymanowski procedure is seldom of value in this type of ectropion. A very effective and simple method is to make a horizontal skin incision three mm. below the lid margin the full width of the lid. The lid skin inferiorly is undermined and allowed to retract. The lid margins are sutured, so the skin defect becomes a wide elipse. This is easily closed with a free full-thickness graft cut by pattern from an upper lid or supraclavicular region.

When sliding or rotating flaps are used, undermining should be very wide to prevent undue tension on the tissue later. The edges are approximated with interrupted sutures of 5-0 and 6-0 silk placed very close together (see color plate, fig. 19, a-h). The graft is covered with dry gauze and pressure maintained for six days with an ace elastic bandage. During this time penicillin is administered daily.

Conclusions

1. Burns of the upper face cause more cosmetic defect than burns of similar extent elsewhere.

2. An almost universal result of these burns, when they are of deep second-degree type or worse, is ectropion of the eyelids.

3. Early treatment of the skin burn should consist of gentle cleaning without debridement followed by vaseline gauze and gentle pressure dressing.

4. If the facial burn is extensive, the granulating area is covered with a split-thickness skin graft as soon as possible, usually in about three weeks.

5. If a third-degree burn is small it may be excised immediately and replaced with a full-thickness graft. This may save many weeks of treatment.

6. Only about 12 percent of patients with burns on the skin of the eyelids actually have burns of the eyeball.

7. When lid conjunctiva is damaged as well as opposing bulbar conjunctiva or cornea, steps must be taken to prevent symblepharon. An immediate mucous-membrane graft is the most effective treatment.

8. Ophthalmic cortisone solution (topical) is of great value in burns of the cornea. After extensive skin burns, systemic cortisone during the first few days reduces the severity of the inflammatory reaction and lessens the tendency for burn shock. ACTH is not indicated in early burn cases. The value of both cortisone and ACTH is dubious in late treatment of skin burns and in healing of grafts.

9. Beta irradiation should be used early on vascularizing leukomas if there is any possibility that ultimately corneal transplantation may be possible.

10. When full-thickness free grafts are used near the lids, small ones may be taken from the upper-lid skin, moderate sized ones from just above the clavicle, and large ones from side of the lower abdomen.

9 Central Street.

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Symposium: Ocular Injuries

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CHEMICAL BURNS OF THE HUMAN CORNEA

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I am sure that each of you has seen a patient on whom you have made the diagnosis of "chemical burn," at the same time doubting, and justly, whether you should consider this case to be a burn and not an irritation from chemical exposure. Unfortunately chemical injuries are relentless in their demand for correct diagnosis.

For practical, not technical, purposes, we define a chemical burn as any exposure to chemicals—solid, liquid, or gas—which results in any damage to the cells of the cornea or conjunctiva. This damage is evidenced by a positive stain by fluorescein and confirmed by examination with the biomicroscope which reveals, under the high-power field, the presence on the cornea of opaque epithelial cells. The conjunctiva does not yield so well to microscopic examination but disturbances of the cells can be identified.

I am not able to argue diagnostic points for I know of no way to determine degrees of burn until such a late date that known therapeutic methods are of no avail. I have learned by experience that injuries from caustics and certain of the complex organic chemical compounds demand prompt diagnosis and meticulous care, even at the expense of treating eyes that should be left untouched. Fortunately, there are no contraindications to this method of treatment and no harm is done to any eye by use of the method.

CLINICAL PROCEDURE

The technique followed in my practice is:

1. FIRST AID

Each employee in a chemical plant is required, in his orientation lectures and discussion with his foremen, to learn that exposure to a chemical should be followed immediately by copiously flushing the eye with ordinary tap water. This may be done by using any of the commercial eye fountains now on the market, or by a simple fountain made by any plant, if it is so constructed that the water is controlled by foot pressure or is a constant stream.

The water from the fountain should bub-

ble up high enough to reach the eye easily and the opening should be capped with soft rubber so that no additional injury is produced by contact with metal. These fountains should be placed at strategic positions about the plant so that they may be easily reached in case of injury.

Five minutes of washing at the fountain is advised and, following this, the patient should proceed at once to the medical dispensary for further care by the plant nurses and doctors, or by specially trained first-aid personnel. No neutralization of the chemical should be attempted, for it has been proven that this logical procedure is unsatisfactory and, in some cases, is actually harmful.

2. Dispensary treatment

The injured eye should be anesthetized at once with a nonnarcotic local anesthetic. I prefer pontocaine because of its hardening effect on the corneal epithelium.

As soon as pain and blepharospasm are controlled, two-percent fluorescein is applied and the eye is flushed with saline. The eye is considered to be an injury in need of treatment if any stain is retained. The eye is then inspected for gross particles of foreign material and any such particles are removed.

Next, washing with a constant stream of saline at room temperature, or better, body temperature, is begun and continued for 15 minutes. The apparatus used is a simple bottle filled with sterile saline of from one- to five-gallon capacity. This bottle is fitted with a syphon and, at a three-foot height above the patient, provides a stream of proper force. A soft rubber tip is desirable to prevent additional injury.

This apparatus has now been given a name in a recent textbook which is, of course, ridiculous. It remains, however, a very usable adaptation of an age-old piece of equipment.

Following the first 15 minutes of washing, stain is reapplied and flushed. If a stain is retained, another 15 minutes of washing is carried out. Stain is reapplied and flushed. This time, if a stain is retained, we designate such an eye as a chemical burn of sufficient importance to be referred at once to the ophthalmologist for care.

It is at this point in the care of chemical burns that I have received unfavorable criticism. I accept this. I will, however, continue to diagnose on the safe side rather than to risk any loss of vision.

Chemicals are being used indiscriminately in the home and other places away from controlled first-aid. Accidents of this type tax the ophthalmologist with additional responsibility, for he must, under these conditions, not only use his ingenuity but he must be prompt in administering treatment.

3. The ophthalmologist's care

Under the standards set up in industry for this injury, the patient who retains stain after 30 minutes' washing has his eye closed with a snug-fitting patch and is referred to the consulting ophthalmologist as an emergency, night or day.

The injured eye should be stained immediately, and a detailed study with the biomicroscope should be made. It is, of course, possible to treat such eyes without the use of the microscopic study, but it seems to me that this sort of "getting by" deserves a designation of carelessness as well as laziness.

The opaque cells seen under the biomicroscope are the cells which have been damaged by the chemical and should be removed at once. Many of the organic chemicals as well as the alkalies, act very slowly on the eye and, although they are seen readily with the microscope, only produce subjective symptoms hours or days after injury.

Due to the arrangement of the corneal epithelial cells, the chemical is adsorbed on the cellular surfaces and slowly penetrates the layers until Bowman's membrane is reached. Injury to Bowman's membrane produces scar tissue, while the epithelium is replaced in kind by the sliding of the remain-

ing healthy cells and by mitosis.

Mechanical removal of the cells which are affected by the chemical seems a perfectly logical procedure and this removal, denuding I call it, is the best and simplest method to convert a chemical burn into a mechanical loss of epithelium, which will heal directly in proportion to the number of cells removed. Unfortunately the presence of infection from internal or external sources modifies this healing process.

Inorganic acid burns are different from the chemical injuries just described and are seen only after all damage is done. They must be denuded and treated according to the actual loss of tissue present.

The best instrument I have found for denuding is a round toothpick wound with dry cotton to form a swab. These swabs are autoclaved for asepsis. The eye is anesthetized with four-percent cocaine. This drug is used because of its softening effect on corneal epithelium which, of course, allows the epithelium to separate more readily.

The swab is applied with sufficient pressure to distort the cornea slightly, wiping with a circular motion from the center to the limbus. The conjunctiva should be wiped in its entirety. Special attention should be given to the upper and lower palpebral conjunctiva and caruncle and lid margins because symblepharon will form if they are insufficiently denuded. All wiping or denuding is done under surgical aspesis.

When the microscopic examination reveals that all the affected cells have been removed, the eye is flushed with stainless merthiolate and closed with antiseptic ointment. Immobilization of the lids with a snugly applied eyepatch is advised. Slight pressure by the patch seems to alleviate some of the pain following a chemical burn.

4. After care

I hesitate to dictate the after treatment for such an eye, since now a chemical burn has been converted into a simple mechanical injury, produced under aseptic conditions. I am sure that all of you have your own treatment for abrasions of the cornea and conjunctiva and I do not wish to say that my treatment is better than yours. However, the routine I follow is to treat the pain with 0.5-percent pontocaine ointment and continue antisepsis with either bacitracin or aureomycin ointment. A snug patch is used until the cornea and conjunctiva are healed.

Healing is usually rapid and complete in 24 to 48 hours, depending, of course, on the extent of the burn and denuding. Clinically, I have found that it is advisable to consider an eye which is unhealed after 48 hours to be an infected ulcer and to institute prompt treatment of foci of infection. Infected teeth have been a common cause of complications in the burned eye.

Mydriatics are *not* used as a preliminary treatment because of the continued impairment of vision in the patients whose injuries heal rapidly. Mydriatics *are* used, usually homatropine first and then atropine, if the complications are persistent. I do not believe, from my experience with several thousand chemical burns, that the chemical penetrates to the anterior chamber in sufficient concentration to be of clinical importance.

If the eye is not healed in 48 hours and the diagnosis of infected ulcer is made, I treat the case daily. Under pontocaine anesthesia the lashes, lids, conjunctiva, and the cornea are carefully cleaned of old secretion and ointment, and the eye is flushed with stainless merthiolate.

Fine methylene-blue powder is applied, a very small quantity, to the unhealed area and flushed at once with saline. A deep stain is retained which penetrates to all minute crevices of the ulcer. The patient must be warned that there will be some additional burning. Then ointment is applied and the eye is closed again with a snug patch.

Recently, I have been using cortisone ointment every four hours and I am sure that it is of definite value in the prevention of anterior-segment involvement. My experience is still limited but I believe that it will

be found to be of value in preventing vascularization of the cornea following extensive ulcers.

Following healing of chemical burns, study with the biomicroscope frequently reveals a superficial corneal opacity which impairs vision. Choline chloride in one-percent solution has been effective in clearing many of these opacities. The drops are used every three hours for as many days as vision improves and, in obstinate cases, for two weeks after improvement ceases, if the vision has not returned to preaccident level.

There will be some doubt in your minds about my use of ointments, due to work published by reputable researchers in our field, but clinically I have found that healing is not materially, if at all, retarded by their use.

In a discussion of chemical burns I could hardly close without mentioning a highly-advertised calsulfhydryl product. Recently I ran a series of cases using this material under as close control as possible in a clinical practice. I found no difference in the healing-time between calsulfhydryl and the method I have just described. There are reports in literature of sensitivity to this material and certainly it produces additional pain, severe in some cases, in an already painful eye. I also believe that it is lacking in antiseptic power and that its use robs the injured eye of the now-accepted benefit of the antibiotics and cortisone.

805 Atlas Building (1).

Symposium: Ocular Injuries

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THE SECONDARY REPAIR OF CHEMICAL AND THERMAL BURNS

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Philadelphia, Pennsylvania

When considering this portion of the symposium one must be prepared to discuss two different possibilities, first those conditions with a lost eyeball possible for correction; and second, those conditions which can develop in the presence of an eyeball with vision—present as such or potentially recoverable. The first requirement is purely cosmetic in its purpose, the second is much more important, however, in that it implies the conservation of vision.

EVALUATING THERAPEUTIC PROCEDURES

In the evaluation of the best therapeutic procedures in any case, it is well to remember that an eyeball may retain good light perception, even some light projection, after perforation of the globe has occurred, either at the time of the injury or later in the course of the recovery period.

Later surgery upon such an eyeball will not be successful if the ocular tension is below the normal, if the anterior chamber remains unformed, or if the eyeball has become changed in size or in shape to an appreciable extent.

The permanent loss of the anterior chamber, if present, is the outstanding bar to successful surgery of the globe. The complication is not uncommon in extensive chemical burns. It is due apparently to the perforation of the anterior segment of the globe by the chemical, the introduction of some of this chemical into the anterior chamber (this can occur without perforation), and, as a result, the formation of adhesions between the iris, the cornea, and the lens. There is much hope for any eyeball in which these complicating anatomic situations are not present, no matter how extensive lid or corneal cicatricial formation may be.

Secondary surgery

An additional admonition as to the secondary repair of such ocular conditions is the selection of the time best for the corrective surgery. This secondary surgery is seldom of an emergency nature (considering the eyeball and its adnexa) but unnecessary delay in proceeding with the correction is not uncommon. Surgery must not be done before all chemical effects have disappeared or subsided. That occurs by the therapeutic neutralization of the chemical or by necrosis of the tissues exposed to the chemical, and the disappearance of this necrosis.

This consideration of best time for further surgical procedures makes it necessary to consider, also, the late treatment of the ocular complications. The final result, in any case, depends to a great extent upon the satisfactory cure of these.

Complications of chemical burns

Iritis is an almost universal accompaniment of chemical burns whenever the cornea has been appreciably damaged. Some of these cases develop a slight to moderate amount of immediate, almost acute, ocular hypertension. Mydriatics must be used, nevertheless, judiciously to be sure but adequately to prevent iris synechias.

Secondary infections

The importance of secondary infection in chemical burns is not too certain because of the value and availability of chemotherapy. Thermal burns, however, have a greater amount of early necrosis and in these a low-grade secondary infection is more common.

Local therapy with the sulfonamide compounds, as an aqueous solution or in an ointment, is satisfactory when indicated; however, these should not be used if the eye continues irritated or if the regeneration of the corneal epithelium, as Hughes stated, "appears retarded."

The use of penicillin, in an aqueous solution, will sterilize the conjunctival cul-de-sacs adequately, even in the presence of extensive necrosis and tissue damage and iontophoresis with sodium sulfadiazine or sodium sulfacetimide, or with such antibiotics as penicillin or aureomycin can be of tremendous value in the treatment of extensive deep corneal and conjunctival ulcerations or abscesses.

It is doubtful whether oncoming symblepharon can be prevented by oil drops, by massage, or by glass rods and comformers. Early mucous membrane grafting is the only certain method of preventing the formation of such lid-globe adhesions or of limiting the extent of these adhesions when they have started.

The use of such grafts plays a tremendous role in the secondary repair of these chemical burns, but equally important—in fact of greater importance—is the use of such grafts in the very early primary treatment of chemical and thermal burns. Their use in thermal burns is basic and is obligatory, as a primary procedure. It frequently has a similar relationship in the treatment of chemical burns.

Ectropion, entropion, contracted socket

If the eyeball has been lost, functionally or by an earlier immediate enucleation, one has an ectropion, an entropion, or a contracted socket to correct, even a combination of these three. Functionally lost globes should be enucleated. Too commonly these are permitted to remain too long, various reasons being advanced—such as, sparing the patient further surgery, using such a globe as the base for a later ocular prosthesis—and with the statement that the retention of this globe can do no harm. All these reasons may be true to a great degree, but they prevent the best possible final cosmetic result. (See later herein.)

Conditions for correction in those cases with a satisfactory globe, actually or potentially, will include entropion, and ectropion as well, symblepharon very frequently, and all degrees of corneal involvement from superficial vascularization and opacification to the development of dense white corneal scars in-

volving the entire extent and thickness of the cornea. Some of these conditions are corrected with ease; others with great difficulty.

Ectropion from tissue destruction must be corrected by the resection of the contracted scar tissue and the use of razor-cut, free-skin grafts. The preparation of the bed for such grafts is important. The essentials are: the resection of all scar tissue, the preservation of all normal subepithelial tissues as fascia and muscle, and absolute hemostasis before the grafts are placed into position (fig.1).

The use of thrombin and the patient's serum, together as a glue for these grafts, is strongly recommended. Certain hemostasis is assured, the grafts are held in accurate approximation with less pressure from the postoperative dressing, and sutures are unnecessary.

Entropion, regardless of the presence or the absence of an eyeball, should never be corrected except with a mucous membrane graft. Occasionally an entropion may be limited wholly to the lower lid and to the lower cul-de-sac with the remainder of the palpebral and bulbar conjunctiva in good condition. If such a case appears, it is permissible to use a conjunctival flap from the superior fornix as illustrated in Figure 2, either alone or in addition to a graft.

A mucous membrane graft is usually necessary for these cases. Figure 3 illustrates such a situation. Such grafts may be needed for the replacement of the entire bulbar and palpebral conjunctival surfaces. It is wise to attempt a complete correction at one operation but several different grafts should be used under these circumstances. Multiple small grafts are easier to obtain from the patient's buccal mucosa and these smaller grafts can be fitted into a correcting pattern easier than the larger grafts.

Figure 3 is in error in that it shows the conjunctival flap in position across the cornea. Neither flap nor mucous membrane should be sutured in this manner. The juxtalimbal edge of each is to be sewn accurately to the sclera at the limbus with an adequate number of fine sutures on an atraumatic needle. A bared cornea resulting from the dissection is permitted to epithelize over by extension of the intact epithelium. Attention to this will prevent an unsightly pseudopterygium.

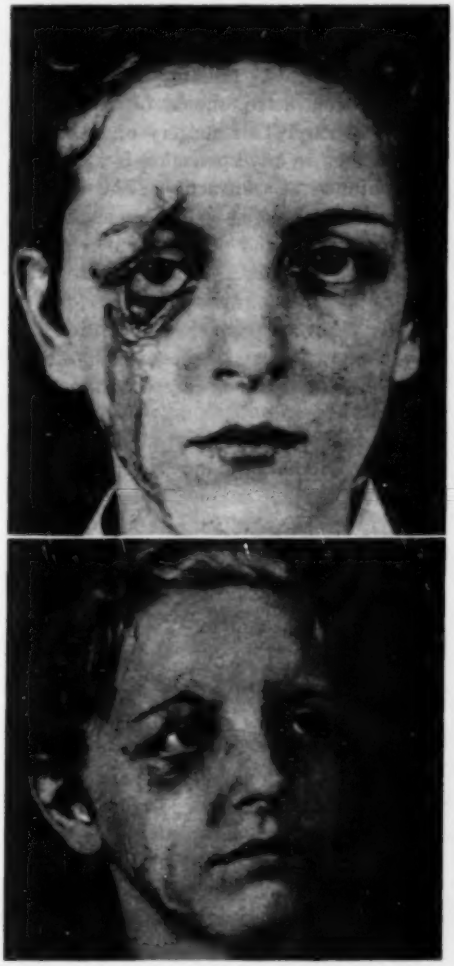

Fig. 1 (Spaeth). Thermal burn ectropion, corrected with free skin graft.

Enucleations

Enucleations, when necessary, are to be done with some type of simple implant into Tenon's capsule—not however of the inte-

grated type. A plastic ball or a bone ball are satisfactory.

All conjunctiva present, even though markedly contracted, should be saved with meticulous care. This surgery must be carried out before any correction is attempted for conjunctival contraction. The residual conjunctiva is closed over the implant in Tenon's capsule and permitted to return to any degree of later contraction before the mucous membrane grafts are used.

After recovery from the enucleation surgery, the grafts may be placed over the exposed capsule, after the resection of conjunctival cicatrices, in any amount sufficient for the correction of any degree of contraction present so that the patient may wear a satisfactory ocular prosthesis. The longer one can postpone a necessary enucleation—if this is not an emergency indication—the better the end result will be from the standpoint of the plastic surgery correction.

Reconstruction of socket

The reconstruction of a socket generally contracted and entirely lost is best done with the use of razor-cut skin grafts. All of the cicatricial tissue in the socket must be re-

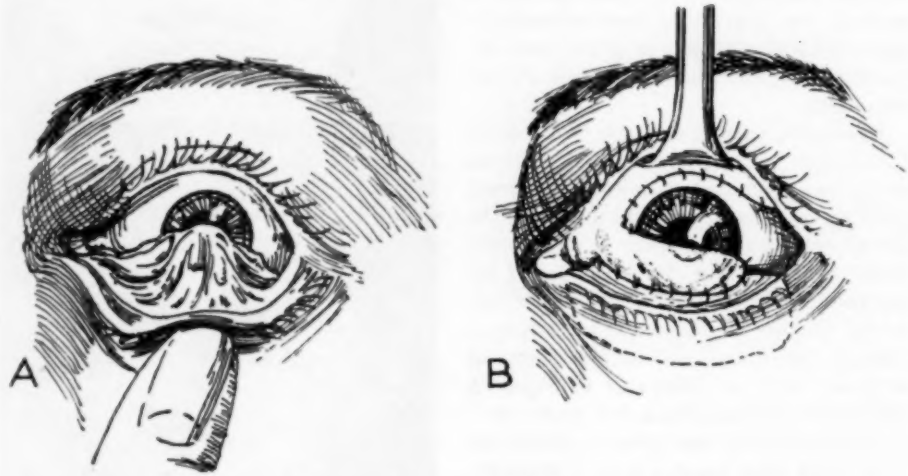

Fig. 2 (Spaeth). Thermal burn (sketch), corrected with pedicle flap of conjunctiva from the superior cul-de-sac.

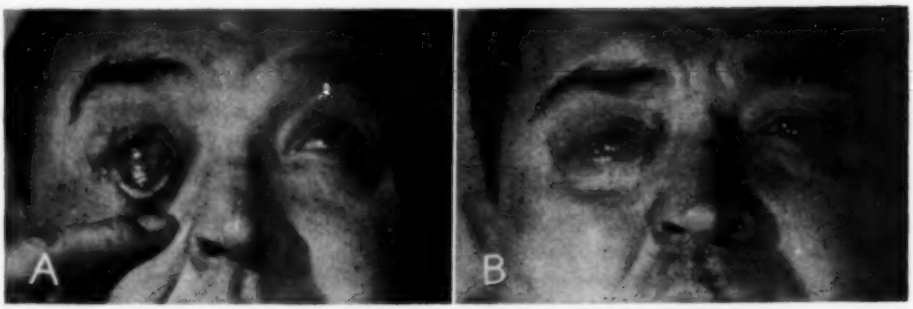

Fig. 3 (Spaeth). Complete restoration of the inferior conjunctival cul-de-sac with a mucous membrane graft. (A) With the lid depressed to show the extent of the graft in the cul-de-sac and over the globe to the level of the inferior limbus and over the cornea for about two millimeters. (B) The patient in repose.

moved until the residual lid tissue is soft and of a normal thickness. Deformed tarsal plates must always be removed, regardless of other conditions. The graft is placed into the dissected site, wrapped about a mold of dental stent, but only after complete hemostasis. Thrombin and autogenous serum are also valuable here.

These cases should not be corrected with a combination of skin and mucous membrane, either one or the other; never together, however.

Radiation therapy

Superficial vascularization and opacification are well treated through the use of radiation therapy. It makes but little difference whether this is administered with radon seeds, with radium-D, or through the use of irradiated salts. The proper use of each preparation is necessary and satisfactory. Occasionally radiation therapy is insufficient and one must use, in addition, actual cautery, with peridectomy, for large deep-lying corneal vessels.

Corneal involvement

Central corneal scars impair vision to a marked degree, disproportionate in degree to the extent of the corneal damage. A small optical iridectomy will improve the visual acuity in these cases, in a most satisfactory manner. Contact lenses also assist very much in such cases in improving visual acuity, as well as giving considerable comfort to the patient.

Perilimbal doughnut-shaped mucous membrane grafts are frequently of great assistance in clearing the superficial cornea of diffuse scars, of superficial vascularization, and in halting the repeated attacks of superficial punctate keratitis.

Surgery indicated

Three types of corneal surgery are to be considered in cases of extensive corneal opacification. These are superficial keratectomy, superficial nonpenetrating keratoplasty, and penetrating keratoplasty. Each of these has rather definite indications.

In general, superficial nonpenetrating keratoplasty can replace superficial keratectomy to a very great extent. If the keratectomy is indicated, a nonpenetrating corneal graft is also indicated and, when successful, will reward with good to very good visual results.

Nonpenetrating keratoplasty

The superficial cornea is carefully removed with sharp dissection, to as great a depth in the cornea as is indicated (including up to two thirds of its thickness), after marking a margin or outline for the graft site with a 10-mm. corneal trephine.

The graft is removed similarly in one piece, without damage to its superficial surface, from the donor eye, using the same corneal trephine as was used for outlining its reception site. This is sutured into position with very fine silk sutures, using traumatic needles, and utilizing at least 16 sutures for accurate approximation. Thrombin and autogenous serum may be used here if desired.

A superficial keratectomy may be done, without an accompanying nonpenetrating graft in cases with a doubtful prognosis because of additional factors other than the corneal opacities, and in instances of superficial but generalized corneal opacification. In these instances a combination of beta irradiation, corneal surgery, and the use of a contact lens may give very good visual results.

Usually a nonpenetrating keratoplasty is a wise procedure. It has another advantage in that it may prepare an eyeball for a later penetrating type of keratoplasty. A case proper for the nonpenetrating type of grafting is usually not a proper case for the use of a penetrating form of corneal graft. It is probably true that the majority of cases are best treated with a superficial graft.

Penetrating keratoplasty

Cases best for treatment with penetrating keratoplasty are those with extensive scarring in the lower portion of the cornea but

with good corneal stroma still present above. The graft must lie in contact with some good cornea if it is to remain transparent.

The corneal scar, in these, is frequently very deep and may be accompanied by iris synechias. An iridectomy can be done, at the time of the grafting, through the area for the reception of the corneal graft. Iris synechias, it seems, influence unfavorably the transparency of the graft. If a cataract is present, or develops later, that can be removed at any time after successful corneal surgery.

Because of the almost certain pathologic change in the curvature of the recipient cornea, it is necessary to suture these corneal grafts into position with many interrupted fine black-silk sutures, exactly the same as one does with a nonpenetrating graft. Twelve to 14 sutures are to be used, placing the suture first at the 12-o'clock position, then at the 6-o'clock position, thereafter at the 3-o'clock and 9-o'clock positions. The remaining sutures are to be placed in between these to obtain a tight and accurate approximation.

Vascularization of the graft does not seem to develop any more frequently in these cases than it does in those with a different etiology. If it does appear, it should be treated by radiation therapy. In general, the utilization of nonpenetrating and of penetrating corneal grafts can be of tremendous value in the treatment of cases with corneal involvement from chemical burns.

1930 Chestnut Street (3).

Symposium: Ocular Injuries

* * *

LACERATIONS OF THE EYE AND ADNEXA*

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Familiarity with the principles of treatment of injuries of the eye and adnexa is essential for anyone concerned with traumatic surgery in either civilian or military life. The ophthalmologist properly should give definitive care because of the specialized techniques which are required, but it is emphasized that the successful treatment of eye injuries, perhaps more frequently than any other, depends upon proper first-aid care. Because the observation of a few fundamental rules in caring for lacerations of the eye and adnexa may mean the difference between preservation and loss of vision, the purpose of this paper is to review the more important principles of both their first-aid and their definitive treatment.

The first-aid care of lacerations of the eyelid should involve sterile dressings and protection of the eyeball, if exposed. Wounds should be carefully cleansed by saline irrigations, if available, and by mechanical removal of obvious foreign bodies. Debridement is usually unnecessary because of the excellent tissue nutrition. As little tissue as possible should be removed and that not unless obviously devitalized.

If the laceration is extensive, the cornea should always be protected either by petrolatum ointment or by the use of a simple suture to pull tissues over the front of the eye. As a rule, antibiotics are unnecessary unless associated intraocular or intracranial injury is suspected. Following such first-aid care, meticulous closure of lacerations should be carried out by someone with some experience in this field.

When confronted by any laceration involv-

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ing the eyelids, care should be taken to exclude the possibility of injury to the eyeball. The ophthalmologist, all too often, sees patients two or three days after suture of what was presumably a simple laceration of the eyelid who had, in addition, a penetrating wound of the eyeball which had become aggravated during the interval.

In addition to inspecting the globe for the possibility of injury, the site of the wound should be examined for possible damage to the orbital walls or margin where a defect might be palpable. Emphysema of the tissues indicates a fracture which has extended into the paranasal sinuses. Enophthalmos suggests fracture of the roof of the maxillary sinus or orbital floor with sinking of the orbital contents.

Unexplained loss of vision may result from a fracture through the apex of the orbit with damage of the optic nerve or hemorrhage into the sheath of the optic nerve. Double vision may result from injury to extraocular muscles or their nerve supply.

Any injury to the lids, particularly if the patient is exposed to high velocity missiles, may be accompanied not only by injury to the paranasal sinuses but by intracranial involvement and for that reason should have adequate roentgen examination following admission to a hospital.

Repair of lacerations of the lid should be done during the first few days after an injury, because defects which are easily repaired with no deformity at the time of injury or shortly thereafter become extremely difficult to correct later and are associated with severe deformity and incapacity. Many instances of prolonged hospitalization for extensive plastic surgery occurred during World War II which could have been avoided by following this simple rule. Furthermore, lacerations of the eyelids rarely become infected and usually heal by primary intention following suturing because of their excellent blood supply.

Early repair is particularly important in treating lacerations through the lid margin because of gaping of the wound and notching of the lid margin which occurs because of the traction of the orbicularis muscle fibers which encircle the palpebral fissure. Shortly after injury, the injury can be repaired readily, but after a week or 10 days the contracted muscle fibers undergo fibrosis and the wound edges can no longer be approximated. If the laceration has been extensive, such severe contracture and fibrosis of the muscle can occur that the severed portions of the eyelid remain only as small nubbins of connective tissue medially and laterally.

Two techniques are available for correcting lacerations through the lid margin. One is the so-called "halving" operation of Wheeler and the other is by simple apposition of the cut edges. In the "halving" operation, a small segment of skin and muscle is excised from one wound edge and a similar amount of tarsus and conjunctival tissue from the opposing edge. The two edges of the laceration then overlap in a latticelike manner when they are sutured.

The other method is simpler and consists only of suturing the severed edges of the wound directly to each other. Interrupted 4-0 mild chromic catgut sutures are placed through the cut edges of the tarsal plate in such a way that when tied the knots are buried within the tissue of the lid (figs. 1-A and B). If necessary, the edges can be freshened and the opposing surfaces made slightly concave by excising a small amount of tissue so that the lid margin tends to pout

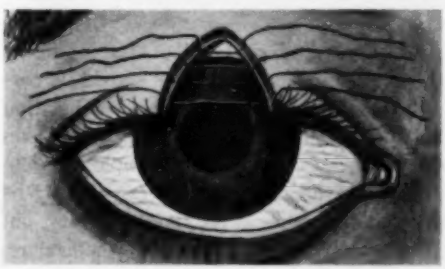

Fig. 1-A (Scheie). Repair of laceration through the lid margin. Deep sutures (4-0 mild chromic catgut) are placed through the tarsal plate.

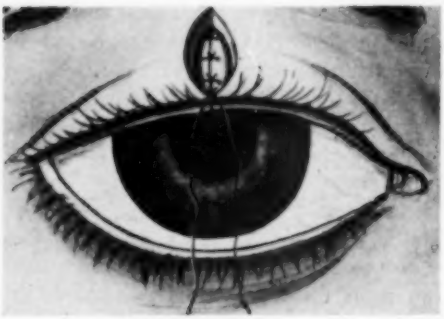

Fig. 1-B (Scheie). Deep sutures tied. Intermarginal silk suture in place.

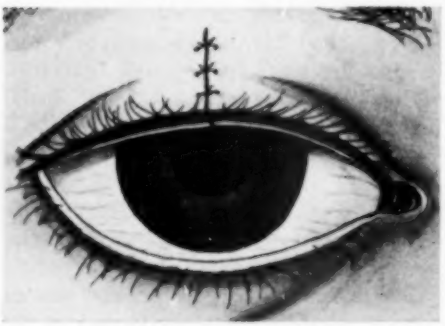

Fig. 1-C (Scheie). Intermarginal suture tied. Skin and orbicularis muscle are closed with interrupted silk sutures.

when the sutures are tied.

After the deep catgut sutures are tied, a silk suture is placed through the intermarginal space at the grayline (fig. 1-B) and, finally, the orbicularis muscle and skin are closed with a row of 4-0 interrupted silk sutures (fig. 1-C). Healing almost invariably occurs and a nice lid margin is obtained.

With loss of portions of the lid, even though extensive, the defects should be corrected as soon as possible by a combination of canthotomy and undermining of skin temporally sufficient to give ample tissue to restore the lid margin. This can usually be done quite readily at the time of injury, but is extremely difficult later after whatever remains of the lid fragments has undergone contracture. If the tissues are under tension, further undermining of tissues should be done together with tarsorraphy causing the upper lid to adhere to the lower.

Reconstruction of lids should be done as well as possible even though the eyeball has been destroyed, because in this way the groundwork is established for providing a socket which will retain a prosthesis. Repair of the lids in instances where the eyeball has been removed is best done over a conformer which aids in establishing a good cul-de-sac.

Tears or lacerations through the canalicular portion of the lid, particularly the lower, are much more serious because of the tearing which follows if repair is unsuccessful. Repair of such injuries is much more difficult than lacerations elsewhere through the lid margin for two reasons:

1. The tissues surrounding the tear are very soft and areolar, being medial to the tarsal plate. The medial canthal ligament is difficult to identify and sutures, when placed, hold very poorly and are usually inadequate to withstand the constant pull of the orbicularis muscle. Notching therefore occurs and tearing is almost inevitable.

2. The canaliculus consists of little more than two layers of epithelial cells and is difficult to suture and reconstruct. Repair over a silver lacrimal probe has been described but, in my experience, this has met with questionable success. A silver probe is bent at one end, introduced through the punctum, passed through the torn ends of the canaliculus, and into the lacrimal sac. The laceration is closed by interrupted deep and superficial sutures and the canaliculus sutured over the probe which is left in place for five or six days. Due to the pull of the orbicularis, however, the wound edges usually retract and the repair is often a failure.

A method which I use and which has proved satisfactory in several instances involves the construction of a new canthal ligament by which the lid is anchored to the periosteum of the anterior lacrimal crest and repair of the torn canaliculus is made over a nylon suture.

First, the torn ends of the canaliculus are

found to be sure they can be identified and repaired. This is usually not difficult with fresh injuries. An incision is made as for dacryocystorhinostomy and the medial wall of the lacrimal sac reflected from the lacrimal fossa (fig. 2-A).

The blunt end of a slightly curved needle, through which a nylon suture has been passed, is then introduced into the punctum, carried through the torn ends of the canaliculus, and into the sac. The needle is pressed medially until one sees the impression of the tip of the needle through the previously exposed medial wall of the lacrimal sac.

The sac wall is slit with a Bard-Parker knife only sufficiently to allow the needle to be pulled through (fig. 2-A). Its sharp end then is passed through the skin just medial to the incision which was made to expose the lacrimal sac after which the needle is cut from the suture leaving two nylon sutures in the canaliculus, one end of each protruding from the punctum, the other through the skin nasally.

The tarsal plate is then exposed through the original laceration and a tongue of tarsal plate is dissected from its lower border in such a manner that it remains attached to the tarsal plate medially (fig. 2-A). The free end of the tongue is then carried medially across the laceration and beneath the skin, which has been undermined, to the anterior lacrimal crest. It is sutured to the periosteum of the crest tightly enough to relieve all tension upon the canalicular portion of the lid (fig. 2-B).

Catgut sutures are then placed to close the conjunctival aspects of the wound (fig. 2-B), and the skin and orbicularis muscle as well as the incision over the lacrimal sac is closed with silk (fig. 2-C). The ends of the nylon suture which run through the canaliculus and lacrimal sac are loosely tied (fig. 2-D) and two or three fine 6-0 silk sutures placed through the torn edges of the canaliculus itself. The canaliculus then usually heals uneventfully over the nylon which is left in place for two weeks. The loop of nylon usually causes only minor annoyance.

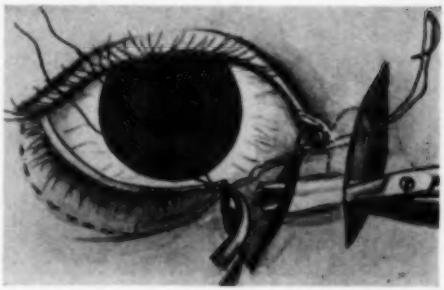

Fig. 2-A (Scheie). Repair of laceration through canaliculus. Incision to expose lacrimal sac. Nylon suture is passed through torn canaliculus and medial wall of lacrimal sac. Tongue is dissected medially from lower border of tarsal plate. Subcutaneous tunnel being prepared through which tongue of tarsal tissue can be passed.

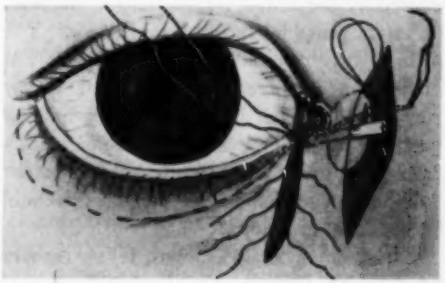

Fig. 2-B (Scheie). Tongue of tarsal tissue is sutured to lacrimal crest. Deep catgut sutures are placed in laceration.

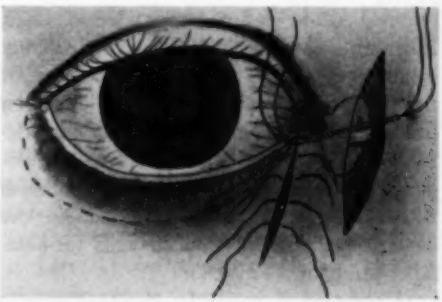

Fig. 2-C (Scheie). Deep sutures tied. Superficial sutures are placed through the skin and orbicularis muscle.

The management of lacerations involving the eyeball itself presents many difficult problems. The immediate treatment, which

Fig. 2-D (Scheie). Final appearance. Ends of nylon suture are tied to form a loop.

is of the utmost importance, is mainly prophylactic, aimed at preventing further injury than that which has already been caused by the initial trauma and to prevent, as far as possible, future complications. The injured eye should be immobilized immediately to prevent herniation of ocular contents. This can be accomplished best by patching both eyes and moving the patient by litter. At times, these conditions cannot be fulfilled but movement and straining should be kept at a minimum.

No attempt should be made to remove what might appear to be foreign bodies or blood clots because of the danger of causing further damage to the eye. Iris tissue may closely resemble a blood clot and attempts at removal could cause irreparable damage. Furthermore, any manipulation of a recently injured eye causes pain with squeezing of the lids and extensive herniation of ocular contents. Such examination should be deferred until it can be done by an ophthalmologist who will employ facial akinesia combined with good anesthesia. In addition, he will have proper lights and instruments for a detailed study of the eye.

If atropine is available, its instillation is indicated in all instances where an ophthalmologist will not be available shortly. Although at times it may have undesirable effects, such as increasing an iris prolapse or promoting anterior synechia, if generalization is to be made, its advantages far outweigh its disadvantages by putting the iris and ciliary body at rest and helping to allay traumatic iridocyclitis.

Chemotherapy or antibiotic therapy should be instituted as soon as possible because infection once started usually causes permanent damage to the eye. A wide choice is available among the sulfonamides, penicillin, streptomycin, and many others. The combination of penicillin and streptomycin has a wide bacterial spectrum and is effective when given in full therapeutic doses. Newer antibiotic substances, such as terramycin and aureomycin, are very satisfactory but may cause gastric irritation and upsets with nausea and vomiting.

Following any laceration which extends into the eyeball, a stimulating dose of tetanus toxoid should be given if the patient has been previously immunized or tetanus antitoxin if not.

The second phase of care, definitive treatment, should involve continuing the above regimen and any of the more specific measures which might be indicated. One's first duty is carefully to examine and evaluate the extent of the injury. The possibility of an intraocular foreign body should always be excluded. This often involves persistent and painstaking effort but failure to do so is one of the commonest errors in caring for ocular injuries. Following these preliminary studies, we are faced with the choice of operative procedure to be carried out.

Several factors influence the choice of definitive surgery, some of which are the type, location, and size of the wound, whether or not the injury penetrates into the eyeball or even through the eyeball, the presence or absence of a foreign body, the presence of lens injury, intraocular hemorrhage, and others. Only a few of the more important of these complicating factors will be taken up. Let us assume first that we are confronted by a patient with a simple corneal laceration, no herniation of tissue or prolapse of iris being involved, and there is no evidence of intraocular injury. Such wounds, if small, can be left untreated except for chemo-

therapy and atropine to put the iris and ciliary body at rest.

If the wound is small and peripheral, miotics may be indicated to prevent iris prolapse or peripheral synechia. If larger, closure can be attempted either in the form of the conjunctival flap, direct suture of the corneal edges with fine atraumatic silk sutures, or a combination of both.

The safer method without excellent suture material is the conjunctival flap. There is always a danger of lens injury while placing direct corneal sutures. This choice is one which should be properly left to the operator for either one is widely accepted.

Scleral wounds are best closed by 6-0 or 7-0 atraumatic interrupted silk sutures. These can remain buried, the conjunctiva being closed over them. No objection can be raised to the use of catgut except that the needles are, as a rule, coarser and more difficult to place, promoting vitreous loss and aggravation of the injury.

Lacerations of the cornea or sclera, when complicated by intraocular damage, are much more difficult to manage. Iris prolapse should, as a rule, be excised and the corneal wound closed as early as is possible. Some surgeons prefer replacing iris prolapses but there is always danger of anterior synechia formation or even recurrence as well as a greater incidence of infection. If the prolapse is associated with lens injury, repair of the iris prolapse should be delayed.

Whenever severe injury of the lens capsule is seen, manifested by signs of a rapidly developing cataract following an injury, operation should be delayed allowing the lens to digest and liquefy. Linear extraction can then be done simultaneously with excision of the iris prolapse and repair of the wound, usually through the same opening. A delay of two or three days usually suffices.

No difficulty will be encountered in freeing the iris from the lips of the wound after the length of time just mentioned and infection ordinarily is not a problem with antibiotic therapy. If, on the other hand, the iris is immediately excised and the wound covered by a conjunctival flap or sutured and the lens allowed to remain, the cataractous lens material becomes highly irritating and predisposes to endophthalmitis phacoanaphylactica, secondary infection, and secondary glaucoma.

It is difficult to bring oneself to attribute these complications and delayed recovery to the irritation of lens material when the clinical picture is so obscured by the trauma of the original injury plus the subsequent operative procedure. One is, therefore, loath to reënter such an eye to perform a linear extraction which, if not done, may well result in calamity.

Although this situation has resulted in the loss of many eyes, the subject is greatly neglected in ophthalmic teaching. Cortisone given systemically, or even locally, seems to offer considerable promise in preventing and treating irritation from lens material and may alter the management of such situations.

In the management of wounds over the ciliary body with or without incarceration of that structure, great judgment is required. The prognosis for the injured eye must always be guarded, although the outcome can be surprising. The threat of sympathetic ophthalmia is always of concern. The best prognosis is afforded by clean wounds with no incarceration of the ciliary body and little or no vitreous hemorrhage.

We must remember that a wound through the ciliary body at right angles to the limbus results in less bleeding because the blood supply runs from front to back in the ciliary processes, the vessels being parallel. A jagged wound or one parallel to the limbus is therefore apt to cause much more bleeding by tearing more vessels.

If visual function is fair as judged by good light perception and projection, wound repair should be attempted even if uveal tissue has prolapsed. The prolapse should be excised or cauterized before placing sutures. Following repair of wounds over the ciliary body, careful observation should be

continued for at least six months.

If the wound is large and jagged, the ciliary body is prolapsed, vitreous hemorrhage is marked, and light perception is absent, prompt enucleation is usually advisable. Emphasis should be made that because there is little danger of shock, hemorrhage, or serious infection, particularly if antibiotics are used, there is no great urgency to operate. In fact, caution against thoughtless enucleation is urged.

The patient should first be examined carefully for evidence of other injury or intracranial involvement. When a patient has suffered ocular injury from high velocity missiles as in war time, no enucleation should be done without first having a skull X-ray study. General anesthesia might mask intracranial bleeding and indirectly result in death. In war time, a fairly innocuous-appearing eye injury often represented the point of entrance of a missile which had lodged within the cranial cavity or passed through the skull leaving a more extensive wound of exit elsewhere.

In many cases, the indications for enucleation are questionable when the patient is first seen and the problem of sympathetic ophthalmia is of great gravity. The ophthalmologist can gain some reassurance from knowing that sympathetic ophthalmia rarely occurs before two weeks following the injury. Eyes in which the indication for enucleation is doubtful can therefore be observed safely for that length of time.

It is of importance that 80 percent of all cases of sympathetic ophthalmia occur within three months of injury and 90 percent by the end of the first year, the most dangerous time being from the fourth to the eighth week. If an eye has been observed for three months, therefore, the danger diminishes rapidly, knowing that 80 percent occur before that time.

Any eye, in which healing of the wound has been interfered with by incarceration of the iris, ciliary body, or lens material or in which there is question of retention of an intraocular foreign body, must, however, be kept under suspicion and carefully observed.

It must also be remembered that not all cases of sympathetic ophthalmia result from injury to the ciliary body but follow incarceration of the iris and even contusion of the eyeball. If an eye under suspicion remains persistently inflamed and begins to soften, it presents the most common exciting cause of sympathetic ophthalmia and enucleation should be done.

Important premonitory signs can be said to exist any time when the opposite eye has become sensitive to light or the patient complains of difficulty in focusing. One of the early manifestations is the appearance of cells in the aqueous of the uninjured eye.

The treatment of sympathetic ophthalmia has been unsatisfactory in the past. Chemotherapy, including penicillin and sulfonamides, is of no value in either its prevention or treatment. Cortisone and ACTH have been reported as showing promise but they cannot be given for indefinite periods of time. The time to enucleate a suspicious eye, therefore, is before pathologic changes occur. When real doubt exists, this should be done by two weeks.

Summary

Certain principles in the first aid and definitive care of lacerations of the eye and adnexa are outlined. Early repair of lacerations of the eyelid is urged to prevent deformity. Immobilization and antibiotics should be utilized for lacerations of the eyeball. The management of iris prolapses, traumatic cataract and wounds over the ciliary body are discussed.

313 South 17th Street (3).

CANTHOPLASTY AND DACRYOCYSTORHINOSTOMY*

IN MALUNITED FRACTURES OF THE MEDIAL WALL OF THE ORBIT

JOHN MARQUIS CONVERSE, M.D., AND BYRON SMITH, M.D.
New York

Malunited fracture of the bones forming the medial wall of the orbit is the result of the backward displacement of comminuted fragments of the nasal bones and the frontal process of the maxilla over the lacrimal and ethmoidal bones beyond the lacrimal sac, thus impinging upon the medial portion of the orbit.

These bony particles may be forced into the ethmoidal labyrinth, causing an out-fracture of its orbital wall, the lacrimal bone and a portion of the lamina papyracea being pushed laterally into the medial portion of the orbital cavity. Early treatment of such fractures has been considered in a recent textbook (Kazanjian and Converse, 1949).

Deformity and functional disturbances depend upon the degree of bony displacement. Deformity affects the fractured nasal framework and the region of the medial canthus (fig. 1-A). The medial canthal ligament is either severed from its bony insertions or displaced laterally with the medial orbital wall.

The laxity of the canthal ligament causes the medial canthus to be pulled laterally, due to a release of tension on orbicularis oculi muscle fibers; the canthus loses its angular shape, becomes rounded, and the caruncle, semilunar fold, and a portion of the sclera may be covered by the laterally displaced tissues of upper and lower lids. Widening of the intercanthal distance occurs.

An evaluation of the extent of the lateral displacement of the deformed canthus can be made by measuring the distance between the midsagittal plane of the face and the unaffected canthus, and comparing it with a similar measurement on the deformed side (fig. 2).

Because the medial palpebral ligament maintains the tautness of the lids against the convex surface of the eyeball, relaxation of the lid margins causes the lacrimal puncta to lose their intimate contact with the eyeball; the mechanism of collection and evacuation of tears is impaired and epiphora results.

More serious, however, from a functional standpoint, is the disturbance of the nasolacrimal apparatus due to obstruction of the nasolacrimal duct by bone fragments which block off the lumen. Stenosis may result from direct laceration of the sac by dis-

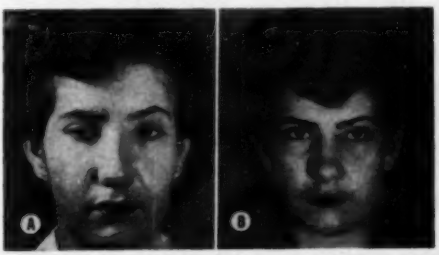

Fig. 1 (Converse and Smith). Deformity of left medial canthal area following comminuted fracture of nasal bones and frontal process of maxilla (Case 3). (A) Shows the typical deformity of the left medial canthus which is displaced forward, laterally, and downward. (B) Result obtained after canthoplasty and dacryocystorhinostomy.

*From the Plastic Surgery Clinic, Manhattan Eye, Ear, and Throat Hospital, and the Plastic Surgery Unit, Department of Surgery, New York University College of Medicine. Read before the New York Academy of Medicine (Section on Ophthalmology) March, 1951, and the American College of Surgeons (Symposium on Injuries of the Eyes), San Francisco, November, 1951.

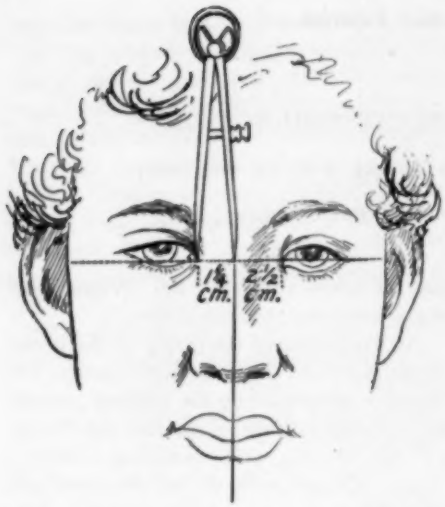

Fig. 2 (Converse and Smith). Caliper test to show the lateral displacement of the medial orbital wall on the left side. The distance between the midsagittal plane of the face and the left medial canthus is elongated.

placed pieces of bone or foreign bodies such as sharp-cutting glass.

Repeated episodes of acute suppurative dacryocystitis tend to follow. A mucocele may form as a result of the chronic inflammation and typical viscous fluid be expressed by pressure over the swollen sac which forms a visible and palpable mass in the canthal region.

More frequently, particularly when the sac itself has been injured, the entire medial canthal region shows thickened, edematous, tender tissues; periodic, acute inflammatory episodes complicate this chronic dacryocystitis, a purulent exudate being discharged by the lacrimal puncta into the palpebral fissure and conjunctival sac.

It is possible to evaluate the extent of the local damage from the history of the accident, the type of injury sustained, and the displacement of bony and soft tissues of the area. In most cases the nasal pyramid is deformed. Various anomalies may be observed in the nasal cavity: deviation of the septum, which presses against the middle turbinate; adhesions between the middle or inferior turbinate and septum; enlargement of the middle turbinate and suppuration in the middle meatus as a result of maxillary, ethmoidal, or frontal sinusitis.

In one of our cases, synechias had obliterated the space between the septum and the entire lateral nasal wall on the affected side. In another case (fig. 3-A) a fragment of an explosive shell was lodged in the anterior portion of the ethmoidal labyrinth and could not be removed until after the anterior tip of the middle turbinate was resected and an anterior ethmoidectomy was done. Achievement of a successful dacryocystorhinostomy is contingent upon the reëstablishment of nasal function.

Clinical confirmation of nasolacrimal-duct obstruction is made when saline, having been introduced by a syringe into a punctum and canaliculus, fails to reach the nasal cavity. Further investigation of the lacrimal apparatus can be achieved with a fine probe after topical anesthesia, using 0.5-percent pontocaine solution; an indication of the size and location of the lacrimal sac or its remnants may thus be obtained.

Clinical examination should be supple-

Fig. 3 (Converse and Smith). A Greek officer wounded in recent fighting showed deformity resulting from comminution by fragment of land mine (Case 1). (A) Note the widening of the left canthal area due to multiple fragments of displaced bone. The soft tissues are thickened due to chronic dacryocystitis. (B) After removal of bone fragment from the left medial canthal area and of fragment of land mine, reattachment of the medial palpebral ligament and dacryocystorhinostomy. The right orbital cavity was obliterated.

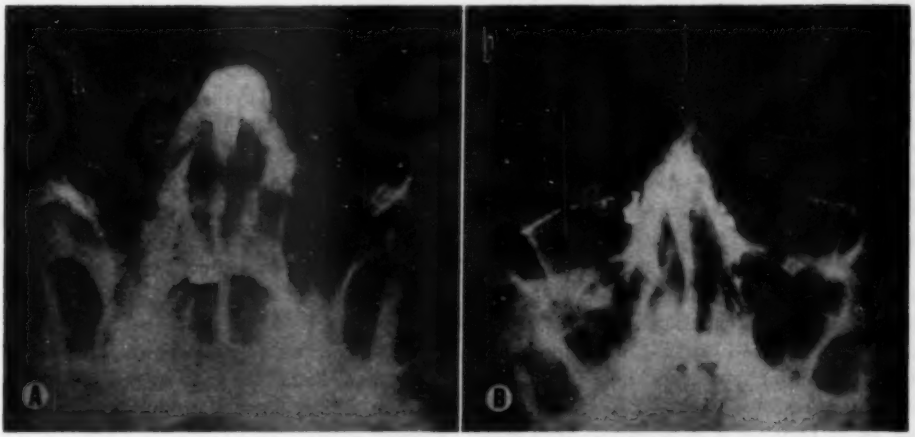

Fig. 4 (Converse and Smith). (A) Roentgenogram of Case 1 (fig. 3-A) taken after lipiodol injection. Shows remnant of left lacrimal sac displaced downward and laterally. Note the lash lines stained by the contrast medium. (B) Roentgenogram of Case 6 after lipiodol injection, in the Waters position, showing the normal left lacrimal apparatus. The contrast medium extends to the inferior meatus of the nasal cavity. It also demonstrates the dilated, obstructed right lacrimal sac. Note also the canaliculi and the lash lines.

mented by stereoscopic radiographic investigation. Displaced bone fragments in the nasal, lacrimal, and ethmoidal areas may be demonstrated by roentgenograms. X-ray examination after lipiodol injection of the lacrimal apparatus reveals the contour of the lacrimal passages or of their remnants (fig. 4-A). In the Waters position, lipiodol may be identified in the lash lines, thus demonstrating the relationships of the palpebral fissure, lacrimal sac, and orbital wall (fig. 4-B).

Combined reconstruction of the medial orbital wall, reattachment of the medial palpebral ligament, and dacryocystorhinostomy

The lacrimal sac is intricately related anatomically to the medial palpebral ligament. The medial palpebral ligament splits into two portions lateral to the sac, enveloping the sac and blending with its walls (fig. 5I-A and B). The anterior portion of the ligament is inserted into the anterior lacrimal crest while the posterior division terminates along the posterior crest of the lacrimal groove. The anterior segment of the ligament is stronger than the posterior but the pull, tensing the eyelids and applying them against the eyeball, is exerted through the posterior portion of the ligament and through the contraction of Horner's muscle.

This small muscle, located immediately behind the ligament, is attached to the bone behind the posterior lacrimal crest (fig. 5I-B). These anatomic facts are important from a surgical point of view.

The reattachment of the ligament and the restoration of the lacrimal apparatus should be completed in the same operation. The ligament should also be reattached to the posterior lacrimal crest, replacing the canthus in its correct position, in order to obtain the posteriorly directed pull normally supplied by Horner's muscle. The rationale for performing the single-stage operation is to avoid the inconveniences of operating in a scarred area at a later stage.

These operations have been performed under local as well as general anesthesia administered through intratracheal intubation. Methylene-blue solution is injected through the lower punctum into the sac; identification

of the stained sac is thus simplified in the course of the operation.

A vertical incision is made over the frontal process of the maxilla, extending through the skin, subcutaneous tissue, and periosteum. This incision should be made anteriorly over the frontal process of the maxilla to avoid the webbing scar which follows an incision made closer to the canthal area.

With a small elevator, the periosteum is raised from the bone along the medial orbital wall over the lacrimal groove and lamina papyracea (fig. 5I-C and D). Malunited bone fragments which protrude into the orbital cavity are resected (fig. 5I-E). The lacrimal sac or its remnant is elevated and the medial wall of the orbit exposed by retracting the orbital contents temporarily (fig. 5I-F).

In some cases the orbital wall is found greatly thickened by overlapping bony fragments; the wall can be thinned by grinding it with a large electrically driven burr to permit adequate replacement of the canthus. The remains of the palpebral ligament are identified anterior to the canaliculus by inserting a small probe into the lower canaliculus through the punctum (fig. 5I-F).

A circular section of bone about 10 mm. in diameter is removed from an area, situated below and anteriorly to the site of the lacrimal groove, by means of a motor-driven trephine (fig. 5I-G); this opening is made without cutting through the nasal mucosa; the trephine osteotomy is enlarged to a diameter of 15 to 20 mm. by rongeurs (fig. 5II-H).

Placing a blunt-tipped probe through the lower punctum into the sac, a vertical "I" incision is established through the wall of the sac (fig. 5II-H); another vertical "I" incision is then made through the nasal mucosa (fig. 5II-I). The anterior tip of the middle turbinate, if hypertrophic, should be resected to prevent obstruction of the pathway to the nasal fossa.

The site of reinsertion of the palpebral ligament is then chosen; it should be at the posterior crest of the lacrimal groove or slightly posterior to it. When normal landmarks are absent, one may be guided by the position of the medial canthus of the unaffected eye.

Two holes are made with a No.-6, round, cutting dental burr, electrically driven, the burr penetrating through the bone but sparing the mucosal lining of the nose (fig. 5II-J). A small curved needle threads fine stainless steel wire through one opening and out of the other (fig. 5II-K and fig. 6-A and B).

A variant to this technique is first to drill a large hole in the bony wall and then to drill another smaller perforation with the No.-6 burr which joins the larger opening at a diagonal (fig. 6-C). The threading of the wire is thus made easier and the stump of the palpebral ligament can be drawn up into the larger hole where it is reimplanted.

The stainless steel wire is looped into the medial palpebral ligament, or the stump of the ligament, and the wire is then held in readiness while proceeding with surgery of

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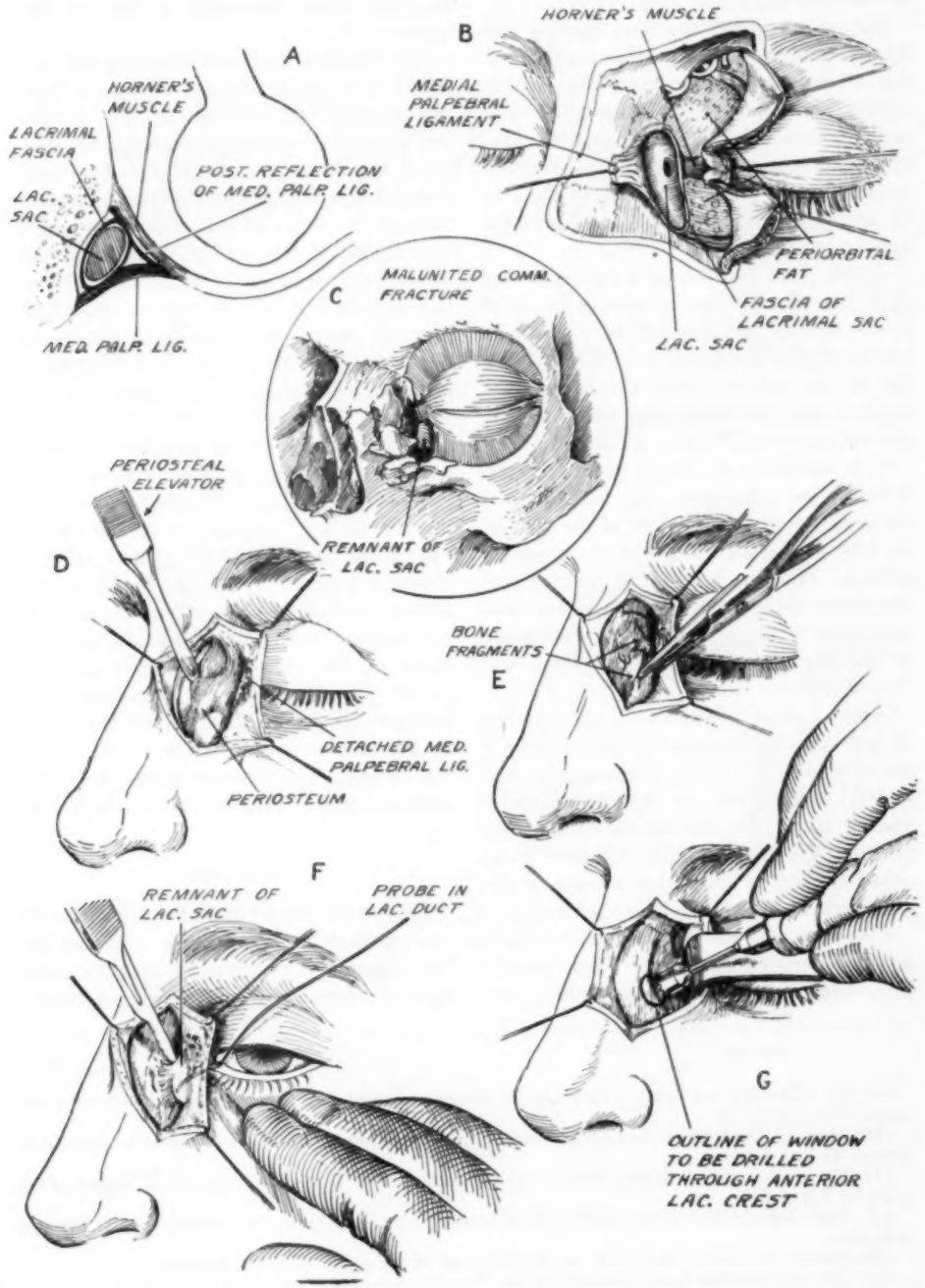
Fig. 5I (Converse and Smith). *Technique of combined canthoplasty and dacryocystorhinostomy.*
(A) Diagram showing the splitting of the medial palpebral ligament into anterior and posterior portions surrounding the lacrimal sac and the position of Horner's muscle.
(B) Diagram showing the relationship of Horner's muscle with the posterior slip of the medial palpebral ligament.
(C) Diagrammatic representation of comminuted bone fragments in the medial portion of the left orbit.
(D) Showing the beginning of the subperiosteal elevation of the medial wall of the orbit.
(E) Resection of protruding overlapping pieces of bone with the rongeur.
(F) Showing placing of probe in the lower canaliculus to help locate the anterior palpebral ligament and define the limits of the sac.
(G) Outline of window to be drilled through anterior lacrimal crest.


A
HORNER'S MUSCLE
LACRIMAL FASCIA
LAC. SAC
POST. REFLECTION OF MED. PALP. LIG.
MED. PALP. LIG.
B
HORNER'S MUSCLE
MEDIAL PALPEBRAL LIGAMENT
PERIORBITAL FAT
FASCIA OF LACRIMAL SAC
LAC. SAC
C
MALUNITED COMM. FRACTURE
REMNANT OF LAC. SAC
D
PERIOSTEAL ELEVATOR
DETACHED MED. PALPEBRAL LIG.
PERIOSTEUM
E
BONE FRAGMENTS
F
REMNANT OF LAC. SAC
PROBE IN LAC. DUCT
G
OUTLINE OF WINDOW TO BE DRILLED THROUGH ANTERIOR LAC. CREST

the lacrimal apparatus (fig. 5II-K).

Having established the bony opening into the nose and prepared the holes for the fixation of the palpebral ligament, fine chromic sutures are placed to approximate the edge of the posterior flap of the sac with the edge of the posterior nasal mucosal flap, and similar sutures are placed through the edges of the anterior flaps (fig. 5II-K and L); these sutures remain untied.

At this point, as the wire is tightened (fig. 5II-L), resistance may sometimes be noted in the area of the floor of the orbit in its medial portion, preventing correct repositioning of the canthus. Scar tissue pulls the angle of the eye downward, resulting in a shortening of the septum orbitale.

It is necessary to dissect downward underneath the orbicularis oculi and to incise the periosteum along the rim of the floor of the orbit to free the attachment of the septum orbitale. The freeing of the scarred and shortened septum orbitale has been described previously in cases requiring reconstruction of the floor of the orbit (Converse and Smith, 1950).

After correct replacement, the canthus should resume its angulated shape and the caruncle should be fully exposed (fig. I-A and B). If difficulty is experienced in replacing the medial canthus in its anatomic position, the lateral canthal ligament is exposed through a small skin incision at the outer orbital margin, and then severed.

One may also cut the tarso-orbital fascia at the outer orbital margin. These procedures, suggested by Wheeler (1939), have not been found necessary in any of our cases.

The stainless steel wire attaching the palpebral ligament to the orbital wall is then twisted upon itself and cut (fig. 5II-L). The fine chromic sutures approximating the posterior flap of the sac to the posterior nasal mucosal flap are tied; similar sutures placed through the edges of the anterior flaps are then tightened. The operation is completed by careful approximation of the edges of the skin incision (fig. 5II-M). An interpalpebral mattress suture maintains the occlusion of the eyelids, and a carefully applied pressure dressing is maintained for a period of five days.

If the remains of the palpebral ligament cannot be identified, or if the canthus itself has been destroyed, it is necessary to place the wire sutures through the medial end of each tarsus, as practiced by Callahan (1950). When a portion of the sac has been destroyed and an insufficient amount of it is left to permit this type of operation, the remnants of the sac, supported on a probe passed through the lower canaliculus, are brought through a nasal window and held by silk sutures passed into the nasal cavity, out through the external nares, and then anchored to the cheek by adhesive tape (fig. 3).

Summary

Deformities of the medial wall of the orbit are repaired by an operation in which the bony contour is restored, the medial canthal ligament reattached, and a dacryocystorhi-

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Fig. 5II (Converse and Smith). *Technique of combined canthoplasty and dacryocystorhinostomy* (continued from Figure 5I).

(H) Showing the I-shaped incision practiced in the sac wall and outline of the bone to be resected to enlarge the long window.

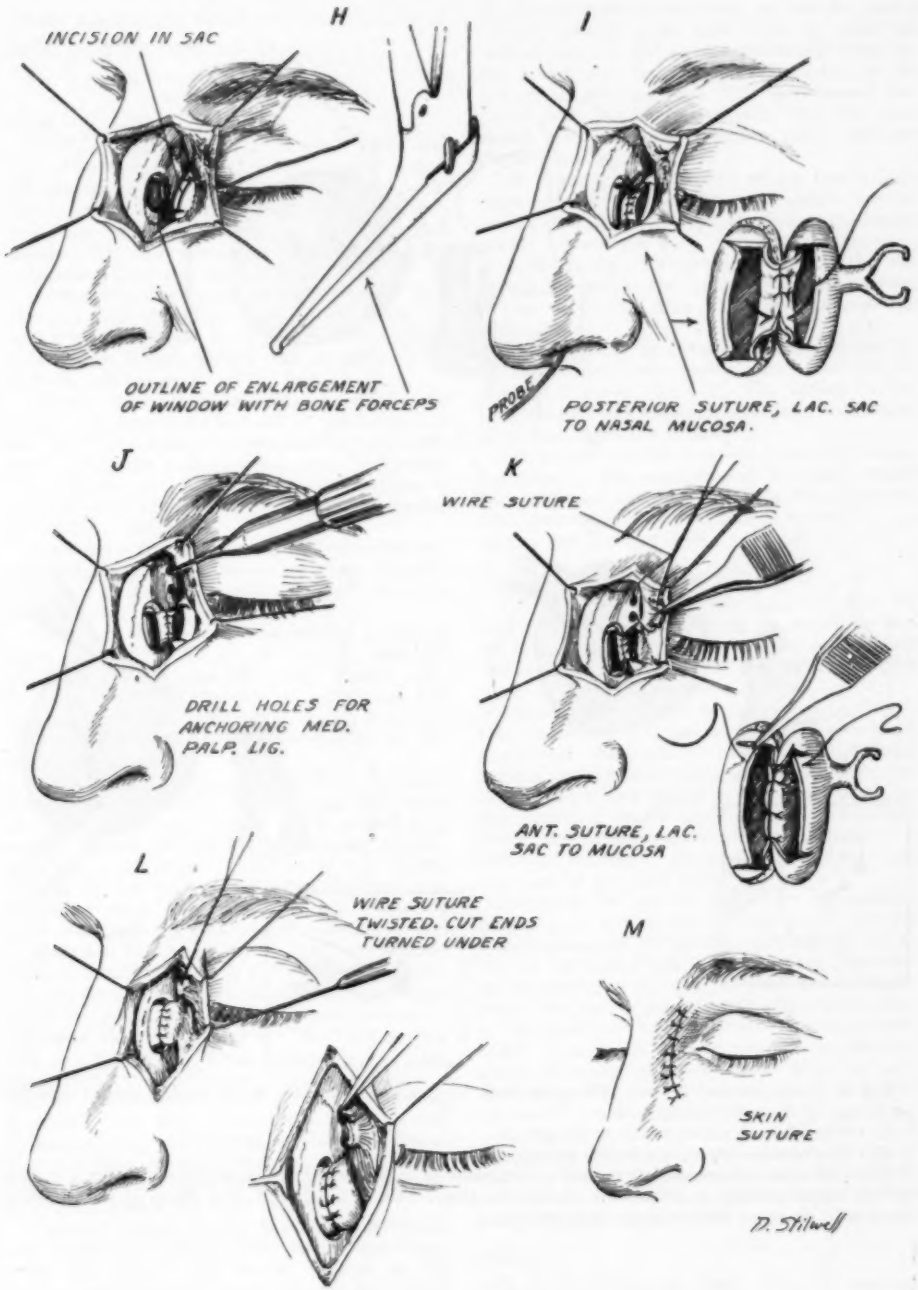
(I) The nasal mucosa has been incised and the posterior flap of the nasal mucosa is sutured to the posterior flap of the sac.

(J) Holes being drilled in the medial orbital wall for the anchorage of the medial palpebral ligament with wire.

(K) Placing of stainless steel wire for the anchorage of the medial palpebral ligament.

(L) The stainless steel wire is twisted and cut. The dacryocystotomy procedure is completed.

(M) Suture of the skin wound completed.


H
INCISION IN SAC
OUTLINE OF ENLARGEMENT OF WINDOW WITH BONE FORCEPS
I
PROBE
POSTERIOR SUTURE, LAC. SAC TO NASAL MUCOSA.
J
DRILL HOLES FOR ANCHORING MED. PALP. LIG.
K
WIRE SUTURE
ANT. SUTURE, LAC. SAC TO MUCOSA
L
WIRE SUTURE TWISTED. CUT ENDS TURNED UNDER
M
SKIN SUTURE
D. Stilwell

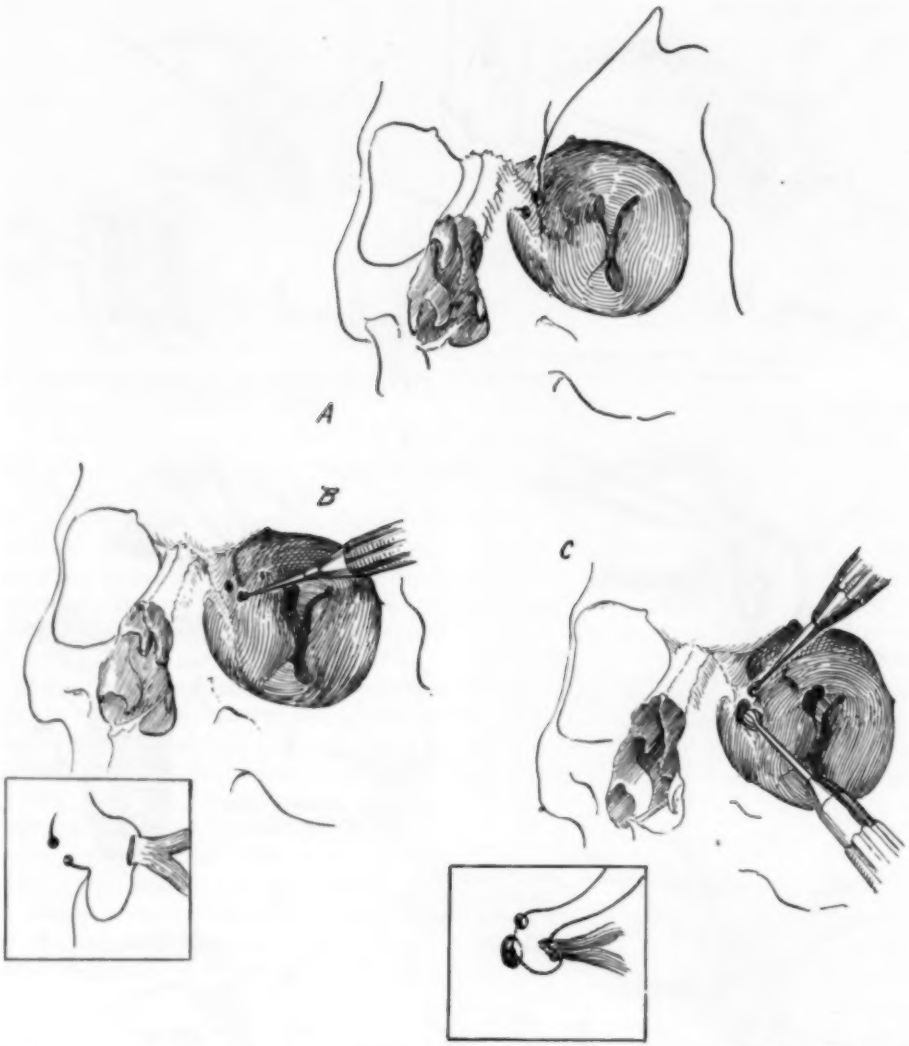


Fig. 6 (Converse and Smith). *Three methods for placing drill-holes in the medial orbital wall for anchorage of the palpebral ligament.*

(A) The holes are placed one above the other.

(B) The holes are placed one behind the other.

(C) One large hole is made at the site of implantation of the medial palpebral ligament. With a smaller drill a second channel is established, joining the larger hole at a diagonal. The stump of the palpebral ligament is drawn up into the larger bony opening.

nostomy is done. Experience in the treatment of six cases is discussed and the histories of these cases are reported.

Case reports

Case 1

V. T., a Greek war veteran, aged 24 years, was injured by an antipersonnel mine in combat against the communist forces on May 18, 1949 (fig. 3-A). The injury to the nose and both eyes resulted in a loss of the right eye, widening of the intercanthal distance, left dacryostenosis, and pansinusitis. The injured right eye was enucleated in Greece.

Examination. He was examined on June 1, 1950, one year after the injury. Examination of the left orbital region showed the typical lateral displacement of the medial canthus with covering of the caruncle and relaxation of the lids.

Marked widening of the distance between the midsagittal plane of the face and the medial canthus was noted. Palpation revealed displaced bony fragments filling the medial canthal area. The patient had epiphora and a history of repeated swelling in the left medial canthal area; the tissue was brawny and tender to palpation.

Intranasal examination revealed adhesions between the septum and the left middle turbinate, obstructing the nasal airway; abundant purulent discharge was present.

Roentgenographic examination showed a piece of steel about 2.0 by 3.0 cm. in the left ethmoidal labyrinth. On April 13, 1950, under general anesthesia, a submucous resection of the septum, left ethmoidectomy, removal of the foreign body, and left radical antrum operation were done by Dr. Richard Bellucci.

Lipiodol study. After reaction from the sinus surgery subsided, lipiodol study showed the source of the left lacrimal infection to be focused in a narrow remnant of the left lacrimal sac situated near the nasal aspect of the lower left orbital margin. The left medial canthus was displaced temporalward about eight mm. The bone fragments nasal to the canthus were irregular and prominent.

Operation. On September 7, 1950, under intratracheal anesthesia, the left medial orbital wall was exposed through a vertical incision, five mm. nasal to the left medial canthus. An aqueous solution of methylene blue was injected into the lower canaliculus to stain the remaining lacrimal passage for the purpose of identification during dissection.

After freeing the adhesions, excessive, thickened, prominent bone was reduced with rongeurs and an electric burr. The lacrimal sac was identified as a narrow tube communicating with the upper and lower canaliculus, of sufficient length to consider connecting it with the nasal cavity.

An osseus window, 10 mm. in diameter, was made with an electric trephine. The nasal mucous membrane was incised, and sutures attached to the lacrimal remnant were passed through the wound and brought out through the left nostril.

Two holes were drilled in the bone above and behind the trephine osteotomy to wire the medial canthal ligament to the bone. After the wire was twisted and cut, the sutures protruding from the left nostril were fixed to the skin surface of the left cheek with adhesive. The skin wound was closed with 5-0 interrupted silk. The lids were closed with a temporary suture and a pressure dressing was applied.

The dressing was removed on the fourth postoperative day. The temporary lid suture was removed. Irrigation through the lower canaliculus readily entered the nose. The skin sutures were removed on the fifth postoperative day. The silk sutures attached to the lacrimal sac were removed on the eighth day. The postoperative course was uneventful.

The position of the canthus was restored, and the lacrimal system has remained free from obstruction and infection (fig. 3-B). This patient has not been troubled with epiphora, sometimes encountered after dacryocystorhinostomy.

The patient was last observed 18 months following surgery. The cosmetic and functional results have remained satisfactory. Since this operation, other procedures have been done to restore the contour of the opposite orbit.

Case 2

J. F., a girl, aged five years, was struck in the face by the door of a moving automobile in November, 1943. The right eye was mutilated and comminuted fractures of the nose and right orbit occurred. The right eye was enucleated.

Examination. She was first examined on July 9, 1951, presenting a typical picture of forward and lateralward displacement of the right medial canthus and depression over the nasal bridge. Despite eight previous operations the condition had not been corrected. The lacrimal sac had been removed. X-ray films confirmed the physical findings of a malunited fracture of the medial wall of the right orbit and of the nasal bones.

Operation. On July 19, 1951, under intratracheal anesthesia, a vertical incision was made over the frontal process of the maxilla; subperiosteal elevation of the medial wall revealed much distortion and thickening of the nasal, lacrimal, and ethmoidal bones. The contour was restored with rongeurs and an electrically driven burr. Two small holes were drilled through the bone to reattach the fibrosed shortened medial canthal ligament. After the wire was twisted and cut, the wound was closed with interrupted 5-0 silk sutures.

A pressure dressing was removed on the sixth postoperative day to remove the sutures. The postoperative course was uneventful and the cosmetic result good. Further procedures for nasal contour restoration are contemplated.

Case 3

M. K., a 19-year-old white, unmarried woman, was injured in an automobile accident July 28, 1950,

suffering multiple lacerations of the face and a compound comminuted fracture of the nasal bones. Emergency treatment was rendered at a nearby hospital; the facial wounds were sutured and the nasal fracture reduced.

Examination. The patient was examined on November 15, 1950. She showed a number of deformities about the face; numerous facial scars, notably a vertical contractile scar extending from the left eyebrow downward across the root of the nose and flatness of the nasal bridge, but the most obvious deformity was the displacement of the left medial canthus covering the caruncle (fig. 1-A).

The patient complained of epiphora and of intermittent attacks of pain, redness, and purulent discharge originating from the medial aspect of the conjunctival sac.

Palpation revealed the presence of bone filling the medial canthal region. Roentgenographic examination showed multiple fractures of the nasal bones, frontal processes of the maxilla and the medial portion of the floor of the orbit. Bony fragments of the nasal bone and frontal process appeared to have been pushed backward over the lacrimal bone and lamina papyracea.

Disruption of the left nasolacrimal system was confirmed when saline solution introduced by a syringe into a punctum and canaliculus failed to reach the nasal cavity; roentgenographic examination after lipiodol injection confirmed the obstruction.

Operation. On December 18, 1950, under intratracheal anesthesia, a vertical incision was made over the frontal process of the maxilla and the bony wall was exposed by subperiosteal elevation. The sac was visualized after injecting methylene-blue solution through the lower canaliculus in order to help identify the structures.

The protruding bone from the medial wall of the orbit was removed with a large-cutting, electrically driven burr until a normal contour was achieved. The remains of the medial palpebral ligament were identified and two holes were drilled in the medial wall of the orbit to anchor the ligament.

A dacryocystorhinostomy procedure was then done and the medial ligament was anchored to the medial orbital wall with stainless steel wire. The wire was twisted and cut and the remaining ends of wire twisted toward the bone. The wound was closed with 5-0 silk sutures.

After placing temporary lid sutures, a pressure dressing was applied. At the end of the first postoperative day, the dressing was so uncomfortable that it was removed for inspection of the cornea.

Although lid sutures were placed, a corneal abrasion was responsible for the discomfort. Whether the abrasion was due to surgical trauma or a displaced cilium was difficult to determine. The pressure dressing was dispensed with and the corneal abrasion was given primary consideration. In 24 hours the abrasion was healed and the patient was comfortable.

On the fifth postoperative day, the skin sutures were removed. The lacrimal duct was patent. Normal contour of the medial canthal region was reestablished and good drainage through the nasolacrimal apparatus was restored, although the patient still complained of slight epiphora when subjected to a strong light. At our last observation on January 2, 1952, the cosmetic result was good, all infection was absent, and the lacrimal passage was functioning (fig. 1-B).

Case 4

H. L., a white youth, aged 19 years, was struck across the bridge of the nose and the right orbital and malar area by the rim of a truck tire, on May 5, 1951.

Examination. The soft tissues over the bridge of the nose and the right supraorbital region were deeply lacerated, the nasal bones were comminuted, as were the right malar bone and the floor of the orbit. Comminution of the floor of the orbit was so marked that collapse of the right orbital contents into the maxillary sinus was observed. The patient complained of diplopia.

Reduction of the fractured fragments was undertaken on May 9, 1951, when the patient was first seen by us, and the maxillary sinus was packed with vaseline gauze to support the comminuted orbital floor. Infection and suppuration occurred despite antibiotic therapy; after surgical drainage, healing occurred, producing an anterior, lateral and inferior displacement of the right medial canthus. A left medial canthoplasty was performed on December 2, 1951.

The lacrimal passage was patent, and care was exercised to avoid disturbing its continuity. Two small holes were drilled into the bone medially, superiorly and posteriorly to replace the medial canthal ligament. The postoperative course was uneventful. The patient was last observed during the fifth postoperative week.

Case 5

D. C., a young white woman aged 19 years, was injured in an automobile collision on May 4, 1949. She sustained an avulsion of the skin of the forehead, a skull fracture, and a deep laceration, nasal to the right medial canthus. The nose was fractured and a laceration extended down across the cheek. An emergency operation was done in Tampa, Florida. The patient was referred to a plastic surgeon in New York who excised and repaired the facial and forehead scars.

Examination. The patient was seen on June 2, 1951. There was a complete ptosis of the right upper lid and a typical downward, forward, and lateral displacement of the right medial canthus. A scar extended from the forehead, across the right median canthus, down to the midportion of the cheek. A skin graft had previously been placed on the forehead (fig. 7-A). Complete obstruction of both canaliculi was noted by probing and irrigation.

Operation. On June 2, 1951, the right medial wall of the orbit was exposed through a vertical incision placed six mm. nasal to the right medial canthus.

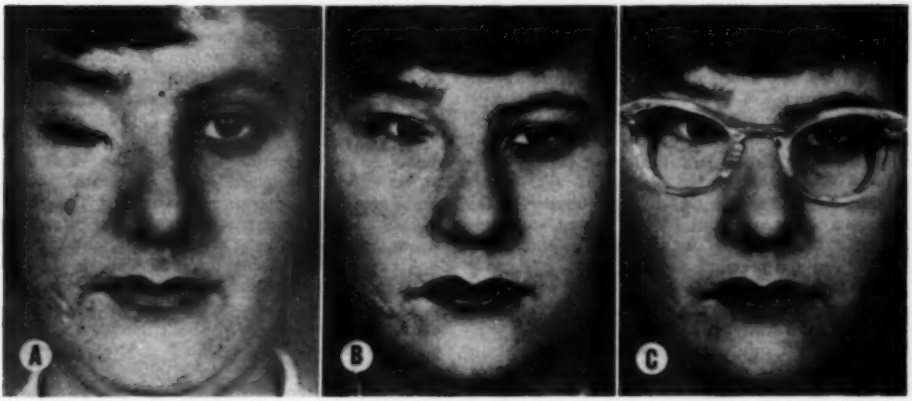


Fig. 7 (Converse and Smith). *Deformity of right medial canthus caused by laceration of the soft tissues and comminuted fracture (Case 5).*

(A) Preoperative view showing displacement of the right medial canthus and traumatic ptosis of the upper lid. The right eye had been enucleated.

(B) Postoperative photograph showing replacement of the right medial canthus, corrected ptosis, artificial eye.

(C) Aspect of patient wearing glasses.

The lacrimal sac had been removed in the course of one of the operations performed before the patient came under our care.

The protruding malunited bone was removed with an electric burr and rongeurs. Two small holes were drilled in the medial wall of the orbit to receive the wire suture attached to the medial canthal ligament. Adhesions, binding the canthus downward and shortening the septum orbitale, were freed from the bone. As the wire was twisted, the medial canthus resumed its normal position.

The skin was closed with 5-0 silk. The lids were sutured and a pressure dressing applied. The sutures were removed on the fifth postoperative day. The position of the canthus was good; the ptosis of the right upper lid, however, was unchanged.

On June 26, 1951, under local anesthesia, the severed levator muscle was identified and sutured to the tarsus, using a conjunctival approach. The postoperative course was uneventful. The cosmetic and function results are good (fig. 7-B and C).

CASE 6

E. S., a young white woman, 20 years of age, sustained an injury to the central, middle one third of the face in an automobile accident on April 21, 1951. Following the injury, a period of unconsciousness lasted two hours. She was treated at an emergency hospital for cerebral concussion, fracture of the nose, and lacerations of the face, nose, and eyelids.

Examination. The patient was first examined by us on September 12, 1951, complaining of deformity of the right medial canthus, epiphora, and a purulent discharge from the right eye. The symptoms had been present and unchanged since recovery from the injury. The vision was normal and there was no disturbance of ocular motility.

The right medial canthus was displaced lateralward and downward in comparison to the normal left side. A purulent discharge was expressed from the puncta upon digital pressure over the right lacrimal sac. Irrigation failed to demonstrate a communication between the obstructed sac and the nose.

Lipiodol studies, shown in Figure 4-B, sustained the physical findings of obstruction of the right nasolacrimal duct.

Operation. On September 17, 1951, under pentothal anesthesia, a vertical incision was made through the skin and subcutaneous tissues down to the periosteum nasal to the right medial canthus. An aqueous solution of methylene blue was injected into the lacrimal sac through the lower canaliculus.

The medial canthus was dissected free from the adhesions binding it to the malunited fragments of underlying bone. The lacrimal sac was identified in a position of downward and backward displacement. The contracted septum orbitale binding the canthus down was severed at its periosteal attachment over the medial one third of the lower orbital rim.

The irregular bone was reduced with a motor-driven burr. Two holes were drilled for reception of the wire attached to the medial canthal ligament. A 10-mm. trephine osteotomy was performed in bone below the drill holes at the site planed for the anastomosis of the lacrimal sac and nasal mucous membrane. The osteotomy was enlarged with rongeurs to a diameter of 18 mm.

Unfortunately, some difficulty was encountered in mobilizing the sac sufficiently to suture the flaps to the nasal mucous membrane without considerable tension; 3-0 chromic catgut sutures were inserted

through the prepared flaps of lacrimal sac and nasal mucous membrane. The wire was twisted and the medial canthus was drawn into a normal position.

The skin and subcutaneous tissues were closed with interrupted 4-0 silk sutures. A temporary interpalpebral suture of silk was placed and tied near the center of the interpalpebral fissure.

On the second postoperative day the patient complained of discomfort in the right eye. Upon prompt removal of the dressing and temporary lid suture, a large abrasion of the cornea was discovered. The abrasion was treated without the presence of the pressure dressing.

Healing was uneventful and all sutures were removed on the fourth postoperative day. The patient was instructed to avoid blowing the nose as a precaution against injury to the wound. On the sixth postoperative day saline solution could be irrigated through the lower canaliculus into the nose with some difficulty.

At the end of two weeks the canthus was in excellent position. However, it was apparent that the dacryocystorhinostomy had failed. It was our assumption that the failure was due to too much tension on the suture lines between the sac and the nasal mucous membrane.

For alleviation of the symptoms of recurrent right lacrimal obstruction, a revision of the dacryocystorhinostomy was done on November 28, 1951. The anesthesia and exposure were identical with that described in the first procedure. The wire suture was located and found to be surrounded by no apparent reaction.

The lacrimal sac was carefully dissected and mobilized well forward. During the dissection, probes were maintained within the canaliculi to avoid injury by the sharp dissection. The nasal mucous membrane extending across the osteotomy was in good condition.

A capital "I" incision was made in the sac and nasal mucous membrane. The anterior and posterior flaps fell into position without tension. The anastomosis was completed with 3-0 chromic catgut and skin was closed with 4-0 silk. A moderate pressure dressing was applied.

Healing was uneventful. On the sixth postoperative day the lacrimal passage irrigated freely with saline. Since that time the passage has remained patent and all symptoms of obstruction have subsided. The patient is pleased with improvement in appearance and restoration of the lacrimal draining mechanism.

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Ophthalmic Miniature

Lagophthalmos is a condition in which the lid is retracted in such a way that it is incapable of covering the bulbus. Ectropion is the condition in which the lower eyelid is everted. Other maladies of the lids are colobomata, loss of substance, and ulceration. Along the edge of the lid occur distichiasis and loss of lashes, moreover the lousy disease, scrofuloderma, hordeolum and, finally, milphosis, in which the lid edges turn the color of litharge.

Aetius of Amida, 550 A.D.
(From the translation by J. Hirschberg.)

OCULAR CONTUSIONS*

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Contusions are injuries which do not cause a breach of the surface structures or an apparent wound, such as those inflicted by blunt instruments or falls. Within such a category one might also class the lesions due to concussion or the remote results of crushing injuries. Payne[7] has recently summarized the effects of ocular contusion.

Contusions may affect the eye and extraocular structures in a variety of ways. The eyeball is protected by the adjacent bony walls on three sides, but on the temporal side a goodly portion is exposed to the effects of direct or indirect injury.

Most contusions in civilian life result from flying missiles, such as the direct blows caused by BB shot, tennis and baseballs, rocks, and so forth. Occasionally, a blow by a fist or a blunt object at the time of an automobile accident causes a nonperforating injury of the eye or adjacent structures.

Contusions as a result of industrial accidents are not too common, and in most instances the injury is produced by a fall against a heavy object. At times a contusive effect is caused by crushing injuries in industry or automobile accidents, and by concussion in underwater explosions or air blasts.

Compared with penetrating and other injuries of the eyeball, contusions are not common, either in civilian or military life. Bellows[1] found only 27 contusions of the eyeball in 500 ocular war injuries, and Rones and Wilder[2] only 26 eyes injured by contusion out of 399 which were enucleated because of injuries. According to my estimate from an article by Thorne[3] contusion injury accounted for not more than six cases of blindness in 367 blinded service men in World War II.

The effect of an injury of this type is determined by the force and direction of the blow. Moderately severe blows directed from an anterior position may cause little distortion or displacement of the eyeball and the resulting injury occurs in the anterior segment.

More severe blows cause distortion of the coats of the eye with disruption of the anterior or posterior structures, or displacement of some of the contents of the eye. If the blow is directed from the lateral side, the eye is displaced nasally against the bony orbit or trochlea and rupture of the coats not infrequently occurs.

Some portions of the eye are vulnerable to injury and may tear; such areas are the limbus, the zone surrounding the optic nerve, the choroid temporal to the disc, the zonules, and the root of the iris. Some of these areas are more susceptible to injury because they are weakened by the entrance or exit of blood vessels and nerves, while others are affected because of the delicacy of the tissue structure and an inability to withstand stress.

Injuries characterized by a sharp impact and short duration usually result in tears and hemorrhage from the internal ocular tissues and some disarrangement of the structures. Other injuries have a contre-coup effect, resulting in a lesion at a point opposite the site of impact.

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Concussion may produce injury to the eye as a result of sudden compression and expansion of the atmosphere, or water. Whether the effect is directly upon the eye or acts through the vascular system has not been determined accurately. It is probable that both act to cause the injuries which have been noted. Rapidly moving projectiles may enter the orbit and cause a concussion type of injury to the ocular tissues without actually coming in contact with the eyeball.

Injuries of the eyelids

The eyelids are covered by a thin skin and possess a lax subcutaneous tissue. Severe swelling is frequent following blows because of this looseness of the tissues. Traumatic ecchymoses are common, and are due to the tearing of the small vessels which lie in the soft tissues of the lid. The hemorrhage occupies the upper lid in the early stages, but it soon settles into the lower lid, and absorbs slowly within one to two weeks. Occasionally the ecchymosis is severe, and an aseptic necrosis occurs in the clot and lid tissues, necessitating an incision and drainage.

Emphysema of the lids with swelling and crepitation on palpation is always a sign of skull fracture affecting one of the nasal accessory sinuses. Usually the air is absorbed and the swelling disappears. The use of antibiotics and sulfonamides may prevent a secondary infection.

Injuries of the conjunctiva

The conjunctiva of the eyeball and fornices is loose and movable, and capable of great swelling as a result of injuries and infections. The portion which lines the eyelids is firmly fixed to the tarsus and is capable of but little swelling. The conjunctiva is vascular and bleeding is not uncommon as a result of an injury.

Ecchymoses of the conjunctiva occur frequently at the time of minor contusions, especially in arteriosclerotic and hypertensive patients. Most minor spontaneous ecchymoses are of no consequence. More severe contusions cause extensive ecchymoses which spread over the eyeball and into the fornices, even causing a prolapse of the mucosa through the lid fissure.

Alarming as some of these ecchymoses may appear they seldom give rise to troublesome sequelae. One must make certain in these instances that the eyeball is not lacerated, or that a foreign body has not entered the orbit or globe. If palpation shows the intraocular pressure to be subnormal, a perforation should be suspected. X-ray pictures should be taken to exclude a foreign body.

Edema of the conjunctiva is a frequent sequel of a contusion, and is often confined to the bulbar conjunctiva. Warm compresses hasten absorption of the fluid.

Injuries to the cornea

The cornea is covered by a thin epithelium which may be dislodged by an oblique blow. Sudden compression of the cornea may result in folding and tears of the deeper portions, imbibition of fluid from the anterior chamber, and prolonged opacification with scarring. The cornea rarely ruptures as a result of a blow, probably because adjacent structures are more vulnerable.

Corneal edema as a result of contusion may be secondary to an epithelial defect, or result from a small tear in the endothelium and Descemet's membrane. The stroma becomes hazy and folds are observed in Descemet's membrane. Rest, bandaging, and mydriatrics usually result in closure of the defect with disappearance of the edema. At times corneal edema may result from lowering of the intraocular pressure. The haziness disappears as the pressure is restored to normal.

Blood staining of the cornea may develop after contusions in which repeated hemorrhages occur into the anterior chamber. The pigment products of the disintegrating red blood cells are said to pass through Descemet's membrane into the stroma where they

lie between the lamellae or in the corneal cells (fig. 1).

Injuries to the limbus and sclera

The limbus is especially vulnerable to contusion injuries because of its special structure. On the inner side it is thinned by the internal scleral sulcus which contains the loose corneoscleral trabeculum. The stroma is further weakened by an extensive plexus of vascular channels. Ruptures of the eye as a result of contusion are therefore not uncommon at this site, and occur concentric with the limbus.

The rupture most commonly occurs in the supero-internal quadrant, possibly because the eye is displaced toward the upper inner orbit and trochlea of the superior oblique muscle. The extent of the rupture depends on the severity of the injury; a portion of the ocular contents may prolapse and be lost.

If the rupture is not accompanied by severe damage to the interior of the eye, the wound may be closed with silk or mild chromic appositional sutures, excising any prolapsed tissue if possible. If the damage is more extensive, the eye should be enucleated.

The sclera is predisposed to injuries at two points: the equator on the temporal side because it is exposed, and in the region of the entrance of the optic nerve to the eye. The perforating posterior ciliary blood vessels weaken the sclera around the optic nerve.

Direct blows may cause a rupture of the equatorial sclera with vitreous loss, and collapse of the eye. The choroidal vessels cause considerable bleeding. If the damage is not too severe, the wound margins may be closed with mild chromic mattress sutures. Rupture of the sclera in the region of the disc as a result of direct contusions is rare, and is usually accompanied by severe intraocular damage. Enucleation is usually indicated.

Injuries to the iris

The iris may be subjected to extensive distortion and displacement as a result of compression by the aqueous humor during a contusion injury. The pupil may be forcibly dilated and stretched, with formation of tears, and the peripheral portion may be displaced backward, causing a separation from the ciliary body.

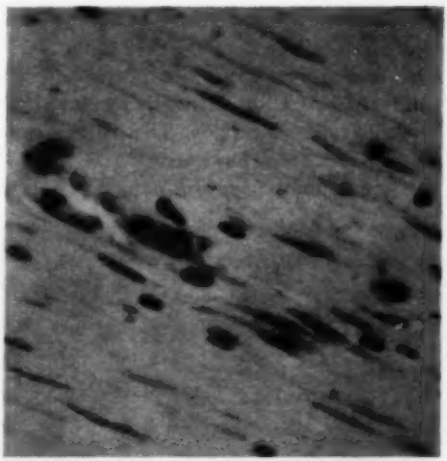

Fig. 1 (Hogan). Blood staining of the cornea. Pigmented phagocytes between lamellae.

Traumatic iridoplegia is a not uncommon result of blows to the eye. Examination shows a large dilated somewhat irregular pupil which reacts poorly or not at all to light. The paralysis occasionally is due to a small tear, but at times is due to concussion with injury to the nerves or sphincter. In many cases complete recovery results, and in others a partially dilated, slightly irregular pupil remains throughout life.

Rupture of the iris is not uncommon. Radial tears are usually seen, especially in the region of the sphincter (fig. 2). They may extend into the periphery, in which case extensive hemorrhage may occur (fig. 3).

Examination shows an irregular dilatation of the pupil at the site of the tear, with an incomplete reaction to light. Slitlamp microscopy reveals the extent of the tear if it is small and localized to the sphincter region. Therapy is not indicated except for bedrest to prevent possible hemorrhage.

Iridodialysis is another common effect of

contusion injuries (fig. 4). The force of the blow stretches the limbal circle and the aqueous humor forces the iris root backward, resulting in separation of the iris from the ciliary body (figs. 5 and 6). The dialysis may be small, or cover a large area.

Examination shows the pupil to be flattened and slightly displaced away from the area of the dialysis. Hemorrhage may or may not accompany the injury. The immediate therapy consists in providing complete bedrest and the use of mydriatics.

After two to three weeks, an attempt may be made to repair the dialysis if it is sufficiently extensive and is not covered by the upper lid. Some authors recommend incarceration of the iris in a small limbal wound. Others incarcerate the iris root and either suture it in place or seal it with diathermy in the limbal wound. The decision as to surgery depends on the patient's symptoms and the cosmetic defect produced by the injury.

Recession of the iris is an uncommon effect of injuries but has been described, especially in the older literature. In these cases the iris is forced backward, probably through a dehiscence in the zonule, against the ciliary body.

Examination shows an apparent absence of the iris at the site of the recession, and the slitlamp shows the iris to be displaced backward. Treatment, except for that of the immediate injury, is not indicated.

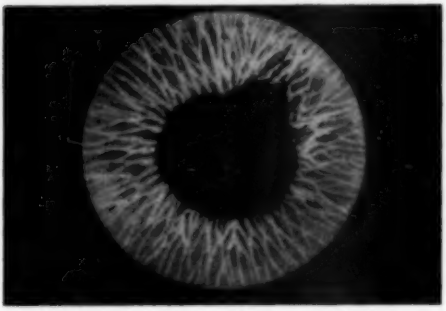

Fig. 2 (Hogan). Rupture of iris through sphincter as a result of contusion.

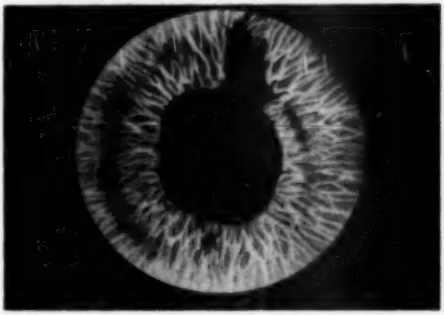

Fig. 3 (Hogan). Complete radial tear through iris due to contusion.

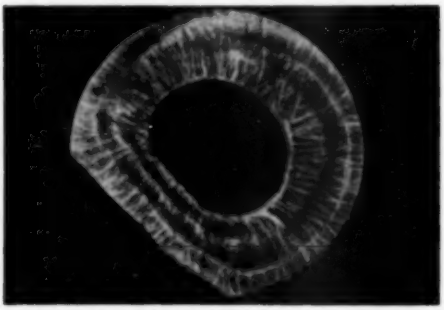

Fig. 4 (Hogan). Iridodialysis due to contusion.

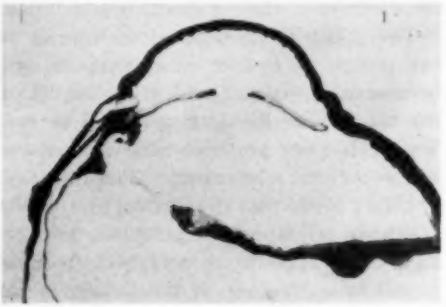

Fig. 5 (Hogan). Traumatic iridodialysis.

Fig. 6 (Hogan). Traumatic iridodialysis.

Injury to the ciliary body

Contusion injuries of the ciliary body may be of serious import because of damage to the ciliary muscle, resulting in accommodative loss; to the vessels, resulting in possible alteration in formation of aqueous humor; and to the nerves which supply the iris and cornea. Contusions are particularly apt to cause lacerations which extend into the stroma near the base of the iris, sever the branches of the major circle of the iris, and cause severe hemorrhage (fig. 7).

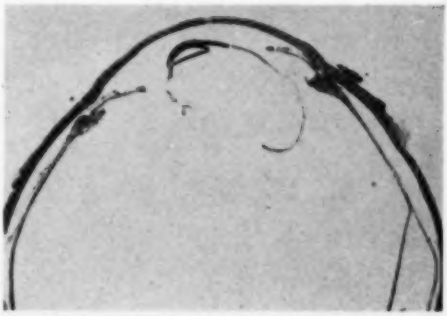

Fig. 7 (Hogan). Hemorrhage into ciliary body (left) and dislocated lens.

Detachment of the ciliary body from the scleral spur is an uncommon injury following contusions, and has the effect of a cyclodialysis. Hemorrhage from perforating vessels over the pars plana may or may not occur. Treatment is restricted to bedrest and use of cycloplegics.

Rupture of the ciliary body is not uncommon, as mentioned above, and bleeding usually occurs into the stroma of the ciliary body and anterior chamber. The hemorrhage usually fills the anterior chamber, following which it gradually absorbs, but may recur on the third to 10th days.

Thygeson and Beard[4] emphasized the seriousness of traumatic hyphema, pointing out that in five of 24 affected eyes the final vision was only light perception or less and that enucleation was required in two cases. All authors who have discussed this subject stress the unfavorable prognosis if a secondary hemorrhage occurs.

Treatment should be directed toward keeping the patient quiet in bed for at least one to two weeks with both eyes bandaged. The occurrence of a secondary hemorrhage into the anterior chamber renders an unfavorable prognosis because of the development of secondary glaucoma and blood staining of the cornea.

Injuries to the choroid

Choroidal tears are a common result of fairly severe contusions of the eyeball. They most frequently occur on the temporal side,

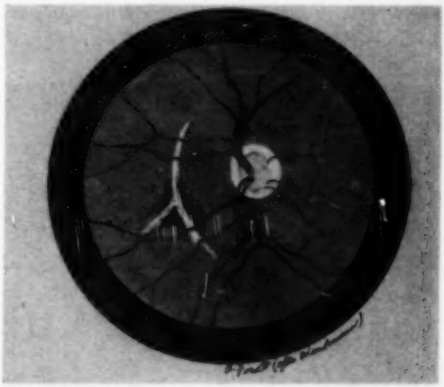

Fig. 8 (Hogan). Choroidal tear of the typical type after healing has occurred. (From Wuerdemann: *Injuries of the Eye*. C. V. Mosby Company.)

concentric with the disc, and between the disc and macula or temporal to the macula (fig. 8). They may be single or multiple. They probably result from stretching of the posterior choroid as a result of compression of the eye, the temporal side being more vulnerable because of its greater extent, and the blow being more commonly directed from the temporal side. It has been postulated that vascular damage to the posterior ciliary channels with resulting choroidal necrosis may cause the lesions.

The tears are crescentic, vertical, and of variable length. Hemorrhage into the choroid, subretinal area, or into the retina frequently accompanies the disruption of tissue (fig. 9).

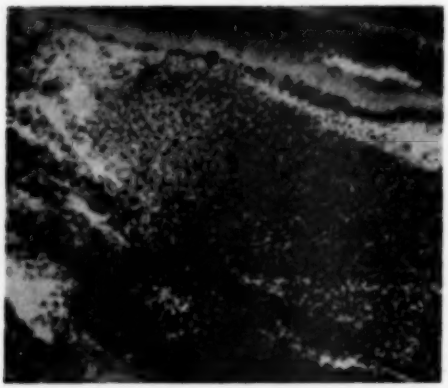

Fig. 9 (Hogan). Choroidal tear with hemorrhage; macular area.

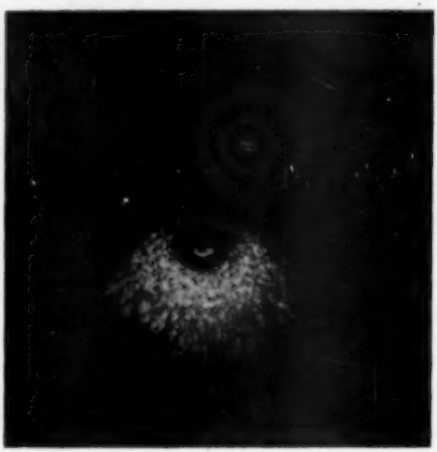

Fig. 10 (Hogan). Macular edema due to contusion. (From Wuerdemann: *Injuries of the Eye.* C. V. Mosby Company.)

Fig. 11 (Hogan). Macular hole.

As the hemorrhage and edema absorb, the yellowish-gray lesions become well defined.

Vision is usually reduced in those instances where the tear lies between the disc and macula, but may be little affected if the tear affects the area temporal to the macula. Treatment is limited to bedrest until the hemorrhage and edema absorb. Tears in other portions of the choroid may occur, principally on the temporal side where the eye is more exposed, accompanied by choroidal, retinal, and even vitreous hemorrhage. Treatment is to advise complete bedrest, cycloplegics, and bandaging the eyes for at least one to two weeks.

Hemorrhage may occur into the choroid as a result of injury, particularly those which are due to concussion. The bleeding may occur around the disc from the posterior ciliary channels or near the site of exit of the vortex veins. The site of bleeding is usually determined by the severity and direction of the blow. Small localized choroidal hemorrhages usually absorb without severe visual defects but larger ones result in retinal degeneration, scarring, and visual loss. The hemorrhage may be sufficiently severe to result in detachment of the choroid.

Injuries to the retina

The peculiar nutritional requirements of the retina, its blood supply, and loose texture make it particularly susceptible to edema as a result of blows. The macular area is most commonly affected. The force is probably transmitted through the ocular fluids which cause sudden compression and possible minute tears.

Following the injury, edema (commotio retinae) occurs, and the macular area exhibits a gray clouding (fig. 10). Vision is usually considerably reduced.

As the edema subsides within one to two weeks, some disturbance in retinal pigmentation is seen, either in the form of a depigmentation or a proliferation of pigment. Occasionally the foveal area undergoes degeneration with hole formation and a retinal

separation may or may not occur (fig. 11).

Experience with crushing and concussion injuries in other portions of the body suggest that vascular spasm may be a prominent factor in the causation of these lesions. The edema may not be confined to the macular region, but may affect a segment supplied by a branch of the central artery.

Treatment must of necessity be restricted to bedrest, quiet, and the use of cycloplegics. The use of vasodilators such as nicotinic acid and papaverine may be valuable in some cases. The prognosis is fair in most cases, vision returning to 20/30 to 20/50; in many cases, however, it is reduced to 20/200.

Rupture of the retina is said to be uncommon as a result of contusions, except at the site of previous degenerative or inflammatory disease. Disinsertions and either tears at the site of previous chorioretinal scars or through areas of peripheral cystic degeneration have been noted (fig. 12). Actual disruption with hemorrhage immediately following a blow is uncommon (fig. 13).

Any injury followed by evidence of retinal damage should be treated by bedrest, bandaging, and cycloplegics until evidence of repair is definite. Retinal detachments and disinsertions should be treated in the usual manner if rest fails to cause a permanent replacement.

Hemorrhages into the retina and adjacent vitreous as a result of contusions are not too uncommon (fig. 14). The hemorrhage usually occupies the area of vitreous adjacent to the injury, or may completely fill the vitreous. In such cases it is impossible to determine if the bleeding has come from the uveal tract until a large amount of blood has absorbed.

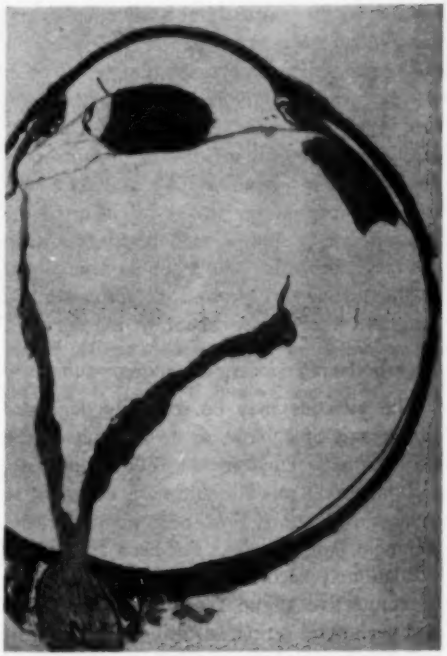

Fig. 12 (Hogan). Oral disinsertion with hemorrhage.

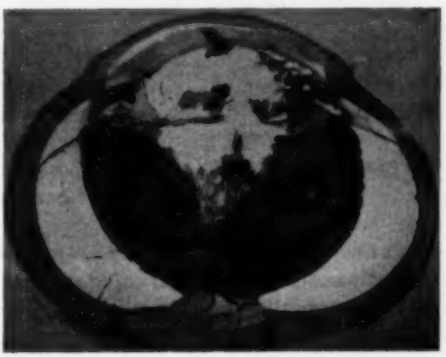

Fig. 13 (Hogan). Retinal and choroidal tears with hemorrhage and detachment.

Injuries to the optic nerve

Optic-nerve injuries are not common following blows to the eye. Papilledema occasionally results as a contre-coup lesion, or may be due to prolonged lowering of the

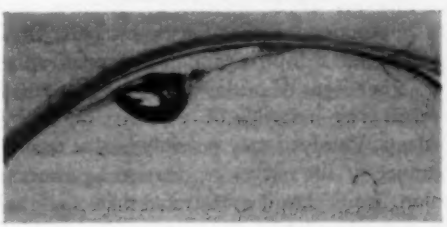

Fig. 14 (Hogan). Retinal and vitreous hemorrhage.

intraocular pressure. If the edema persists, damage to the nerve fibers may result, and the visual acuity is correspondingly affected.

Hemorrhages into the disc or nerve structure may follow an injury to the smaller capillaries. Rupture of the lamina cribrosa due to sudden distention of the posterior segment may be accompanied by hemorrhage and marked visual reduction. Occasionally the optic nerve is torn from its attachment to the sclera and choroid, resulting in the condition known as evulsion of the nerve.

Injuries to the vitreous

The vitreous may be seriously deranged as a result of a blow, or as a result of the outpouring of edema fluid, cellular exudate, and hemorrhage. The ultimate effect is to cause shrinkage of this structure with a detachment from the disc. The hemorrhage and exudate may become organized by extension of connective tissue from the ciliary body and peripheral retina, resulting in permanent opacification and corresponding reduction in visual acuity.

Sommerville-Large[6] found that 23 percent (112 eyes) of 443 eyes injured in the Burma campaign suffered concussion injuries. Of these 43 eyes eventually retained useful vision (6/60 or better); 26 eyes ended with an organized vitreous due to hemorrhage; 23 had gross vitreous opacities; 18 had choroidal ruptures; and two cases had detached retinas.

If an extensive and slowly absorbing hemorrhage is present, good light projection retained, and the other injuries do not preclude a surgical procedure, a vitreous replacement should be considered to prevent organization of the clot.

Injuries to the lens

Spasm or paralysis of accommodation may follow minor injuries and be accompanied by no other signs of ocular damage. Spasm may result from irritation of the ciliary nerves by edema. Paralysis may possibly be due to severance of the nerves or damage by concussion. Smith[5] has demonstrated that minor atmospheric concussion blasts may produce accommodation deficiency as well as varying degrees of retinal edema.

Spontaneous recovery occurs in most cases within one to two weeks. Astigmatism may be produced by actual distortion of the lens by the blow, slight subluxation of the lens, or swelling due to the injury. Small zonular tears may cause a permanent lenticular astigmatism.

Direct blows to the eye cause an increase in the pressure of the aqueous against the lens, with rupture of the zonule, and resultant subluxation or luxation. In addition to the visual disturbance iridodonesis is present on movements of the eye and the anterior chamber may be deeper than in the fellow eye. If examination permits, the luxated lens can be seen in some quadrant of the vitreous, possibly still held in place by some zonular fibers.

Treatment is not indicated in cases of luxation if other complications are absent. Glaucoma occasionally results and may necessitate removal of the lens with a loop, but the results are usually poor. Treatment of the glaucoma should more likely be directed toward providing filtration, than in attempted removal of the lens. Treatment is not indicated for subluxation, except in the case of development of cataract or glaucoma, when an extraction may be done.

Opacification of the lens is seen in several forms following blows to the eye:

1. The Vossius ring opacity most commonly occurs after blows to the cornea. In many cases an intraocular hemorrhage occurs. The ring opacity is of the "colorless" type or the "colored" type and varies from two to three mm. in diameter. It lies on the anterior capsule, and is probably due to a deposition of blood pigment on the lens capsule at the termination of the zonular lamella near the anterior pole (fig. 15). The "colorless" type is probably due to damage to the lens epithelium or underlying cortical fibers and may or may not result in a permanent opacity.

2. Posterior and anterior subcapsular cataracts may develop due to rupture of the capsule or by derangement of the lens fibers (fig. 16). At times the opacification is reversible and at other times it proceeds to complete cataract formation which may require surgical removal (figs. 17 and 18).

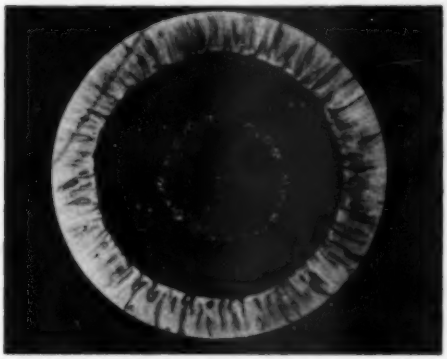

Fig. 15 (Hogan). Vossious ring opacity of lens due to trauma. (From Wuerdemann: *Injuries of the Eye.* C. V. Mosby Company.)

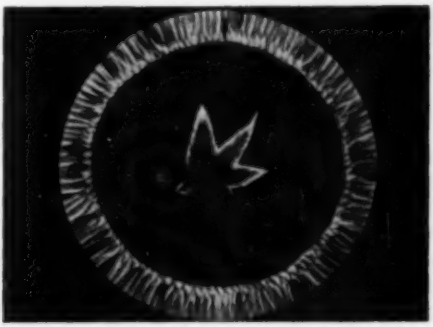

Fig. 16 (Hogan). Anterior subcapsular traumatic cataract due to contusion. (From Wuerdemann: *Injuries of the Eye.*, C. V. Mosby Company.)

Fig. 17 (Hogan). Traumatic cataract.

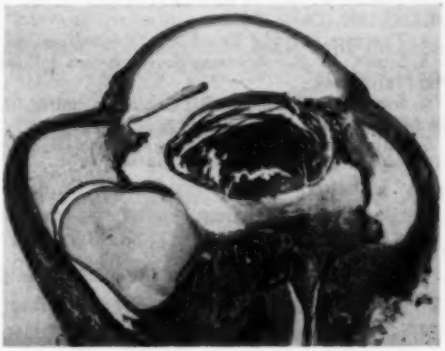

Fig. 18 (Hogan). Traumatic cataract.

Alterations in intraocular pressure due to contusion

The intraocular pressure may be lowered by severe contusions due to damage to the ciliary body. In most cases it returns to normal as the damage is repaired, and vascular tone restored.

The pressure may be elevated by hemorrhage, dislocation of the lens, and iridocyclitis. Treatment should be expectant until the acute phases of the injury have subsided. If the increased pressure persists after one to two weeks a surgical procedure may become necessary. Cortisone applied topically may have an ameliorating effect on the iridocyclitis, and favor restoration of a normal pressure.

Injuries to the orbit

Aside from the injuries caused by fractures of the adjacent orbital bones, contusions of the head may be accompanied by intraorbital hemorrhage and proptosis. The hemorrhage most often gradually absorbs and the eye returns to its normal position.

The extraocular muscles may suffer various forms of paralysis as a result of contusion injuries. The pulley of the superior oblique muscle may be detached as a result of a blow over the eye. The paralysis may be due to hemorrhage into the muscle or around the nerve supplying the muscle. Recovery is the rule in those cases without coincident fracture or intracranial injury. Those cases having a detachment of the oblique pulley

may require a surgical procedure to effect its replacement.

Conclusions

1. Ocular contusions in civil life result from flying missiles, such as the direct blows caused by BB shot, tennis and baseballs, rocks, and toys. In military life the contusions may be due to similar causes, and in actual warfare flying missiles may cause contusions by the kinetic energy developed by the foreign body in passing through the orbit. Concussion blast of the air or water type may also cause ocular contusion by the sudden compression and rarefaction of the air or water waves.

2. The most common effects of contusion are sphincterian or radial tears of the iris, iridodialysis, lacerations of the ciliary body with hemorrhage, subluxation or dislocation of the lens, retinal edema, and choroidal tears.

3. Treatment of most of these lesions is confined to bedrest, employment of cycloplegics, and bandaging the eyes until the danger of hemorrhage is past. Recurrent hemorrhage from a tear in the iris or ciliary body is a serious occurrence and presages either secondary glaucoma with loss of the eye, visual loss due to retinal detachment, blood staining of the cornea, or membrane formation in the pupillary region.

450 Sutter Street (8).

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Ophthalmic Miniature

There is hope if the cataract is small, and immobile, if it has also the color of sea water or of glistening steel, and if at the side there persists some sensation to a flash of light. . . . The case is worse when the cataract has arisen from a severe disease, from severe pains in the head or from a blow of a violent kind. . . . Neither a small nor a sunken eye is satisfactory for treatment. And in the cataract itself, there is a certain development. Therefore we must wait until it is no longer fluid, but appears to have coalesced to some form of hardness. . . . The needle is to be sloped against the suffusion itself and should gently rotate there, and little by little guide it below the region of the pupil; when the cataract has passed below the pupil it is pressed upon more firmly in order that it may settle below. If it sticks there the cure is accomplished.

Aurelius Cornelius Celsus, *De Medicina*, 30 A.D.
(From the translation by W. G. Spencer.)

INTRAOCULAR PENETRATION OF CORTISONE AND ITS EFFECTIVENESS AGAINST EXPERIMENTAL CORNEAL BURNS*

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The present study was undertaken to determine the value of cortisone in the therapy of corneal burns. The study was divided into two main phases: (1) The intraocular penetration of locally and systemically administered cortisone; (2) the actual trial of each method of therapy against standard corneal burns.

METHODS

PENETRATION STUDIES

The aqueous humor of 10 normal rabbit eyes was analyzed for substances giving the cortisone color reaction to phenylhydrazine. The test was devised by Porter and Silber. The vitreous humor of 10 normal rabbit eyes was similarly analyzed.

Aqueous specimens were withdrawn through a 25-gauge needle and tuberculin syringe. The eyes had been anesthetized previously by local tetracaine hydrochloride (1.0 percent). They were thoroughly flushed with normal saline before withdrawing the aqueous-humor specimens. Vitreous-humor specimens were obtained following the use of local anesthesia and flushing, using a 19-gauge needle.

Topical instillations. Cortisone-acetate suspension diluted 1:4 in zephiran (1:10,000) was instilled into the eyes of 25 rabbits. One drop was instilled every hour for six doses, and then every two hours for four more doses. This was continued for four days, or 96 hours, from the onset of therapy.

Aqueous humor was withdrawn at 24, 48, 72, 96, and 120 hours. Eight to 10 eyes were used for each time interval. Vitreous specimens were withdrawn from the same eyes at the same time.

Analyses were run using the Porter-Silber reaction. This experiment was repeated using cortisone-acetate suspension containing 25 mg. of cortisone per cc. of saline. No wetting agent vehicle was employed. Local application was made similarly for four days.

Subconjunctival injection. Cortisone acetate, 1.25 mg., was injected subconjunctivally twice daily for 48 hours, that is, one injection every 12 hours, four times; 1.25 mg. were given once daily for the next two days. Ten rabbit eyes were used for each time interval. Subconjunctival injections of 12.5 mg. and of 25 mg. were made daily for three days. Six eyes were used for each time interval.

Analyses were run at 24, 48, 72, 96, and 120 hours after the initial injection. No injections of subconjunctival cortisone were given after the fourth day. Aqueous and vitreous specimens were withdrawn and analyzed as described.

Retrobulbar injection. Twenty-five mg. cortisone-acetate suspension (1.0 cc.) were injection retrobulbarly once daily for three days. Six eyes were used for each time interval. Analyses were run on aqueous and vitreous specimens obtained at 24, 48, 72, 96, and 120 hours after the initial injection.

Systemic administration. One hundred mg. cortisone-acetate were administered three times the first day at eight-hour intervals; two times the next day at 12-hour intervals, and for the next two days one daily injection was given. Five rabbits (10 eyes) were used for each time interval. No injections were given after the fourth day.

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Aqueous and vitreous-humor analyses were made at 24, 48, 72, 96, and 120 hours after the initial injection.

Corticotropin (ACTH) was administered as 20 mg. intramuscularly four times a day for two and one-half days. Eight eyes were used for analyses at each time interval. Analyses were determined at 24, 48, 72, and 96 hours after the initial injection.

Production of corneal burns

Three types of burns were induced on the corneas of normal rabbit eyes—alkali, acid, and thermal. Before burning, the eyes were anesthetized with three drops of 0.5-percent tetracaine hydrochloride.

Alkali burns

Alkali burns were produced in locally anesthetized eyes. Concentrated sodium hydroxide was used in its solid form. The pellet was grasped with forceps and its narrow margin was applied to the cornea in a vertical position within the limbus on the side away from the nictitating membrane. The pellet was held to the cornea for 10 seconds. Five minutes after burning, both eyes were flushed copiously with normal saline.

Thermal burns

In eyes locally anesthetized with 0.5-percent tetracaine hydrochloride, an electrocautery was applied to the cornea. The cautery was kept at a constant setting and touched superficially to the cornea in three adjacent areas to form a vertical line not involving the limbus and on the side away from the nictitating membrane.

Acid burns

Acid burns were produced with 70-percent sulfuric acid. An iris spatula, bent at right angles so that 2.0 mm. by 3.0 mm. could be applied to the corneal surface, was dipped into sulfuric acid and applied to the corneal surface for 10 seconds. Each eye was flushed copiously with normal saline five minutes after the eyes were exposed to the acid. The burns did not involve the limbal area and were on the side of the cornea away from the nictitating membrane.

Ninety rabbits were used in producing these corneal burns. Thirty were used for each type of burn—that is, 60 eyes were burned with acid, 60 eyes with alkali, and 60 eyes thermally. In addition to the flushing for acid and alkali burns, therapy consisted of the following:

Ten eyes of each group were treated with local cortisone suspension, 1:4 in zephiran (1:10,000). One drop was instilled every hour for six hours; one drop every two hours for three more doses each day. Therapy was continued for five days. In each instance, the right eye was treated and the left eye was the control and received saline drops only, at the same time intervals. Drop therapy was started 15 minutes after burning.

Ten rabbits of each group were treated with cortisone acetate subconjunctivally: 1.25 mg. were injected subconjunctivally twice a day for the first two days, and then once a day for three additional days. In this group, again, the right eyes were treated and the left eyes were untreated. Subconjunctival injections were started one hour after burning.

The third group of 10 rabbits in each series was treated with cortisone acetate intramuscularly: 100 mg. were injected three times a day for the first 24 hours, at eight-hour intervals; 100 mg. for the next 24 hours at 12-hour intervals; and 100 mg. daily for the next three days. Intramuscular therapy was started one hour after burning.

Results

Penetration studies

Pentration of topically administered cortisone. The results of the analyses of normal aqueous humor and of the aqueous and vitreous humor of eyes which received cortisone-acetate suspension as drops can be seen in Figure 1. Each determination is plotted on the graph.

It is evident that as therapy is continued from day to day, the concentrations of detectable cortisone increase in the aqueous humor. By 72 hours, that is, after three days of continued therapy, a peak level is obtained in the aqueous humor. Although therapy was continued for another day, it did not exceed this level. A plateau was reached. With cessation of therapy, after four days of treatment, an immediate drop occurred in the concentration of cortisone in the aqueous humor.

It is apparent that drop therapy with cortisone-acetate suspension produces a detectable amount of cortisone acetate in the vitreous humor. The peak level is arrived at more slowly than in the aqueous and never reaches the concentration obtained in the aqueous humor. The cortisone disappeared from the vitreous humor at a slower rate than from the aqueous humor.

Higher concentrations were obtained with the preparation containing 8.0 mg. of cortisone suspended in zephiran than after use of 25 mg. suspended in saline. (See Figure 2.)

Penetration of subconjunctivally administered cortisone. According to the results plotted in Figure 1, it is evident that subconjunctivally administered cortisone produced a higher average level than did topically administered cortisone. The maximal level reached was obtained within 24 to 48 hours. The peak was reached more rapidly than by drop instillation. When the number of injections were reduced to one per day, the aqueous-humor concentration dropped off quickly and, with the cessation of therapy, a precipitous fall occurred in the aqueous concentration within the succeeding 24 hours (figs. 1 and 3).

The vitreous humor showed a greater concentration following subconjunctival injection than following drop instillation. With

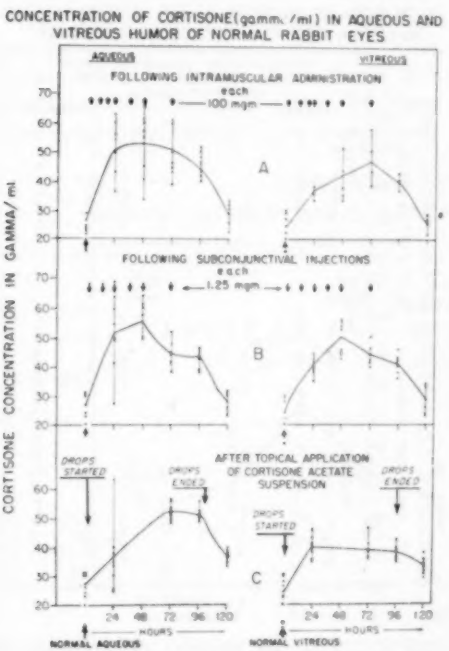

Fig. 1 (Leopold and Maylath). Aqueous and vitreous analyses.

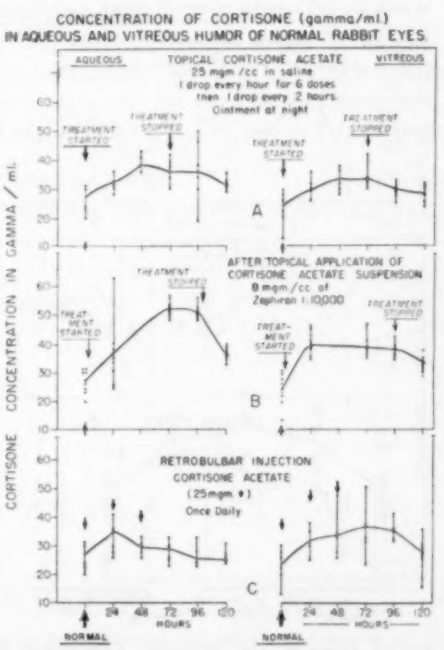

Fig. 2 (Leopold and Maylath). Comparison of zephiran and saline suspensions of cortisone.

reduction in the number of injections, the vitreous levels fell, and when therapy was stopped the vitreous concentrations decreased rapidly but not as precipitously as the aqueous-humor levels.

Increasing the concentrations injected subconjunctivally did not increase the intraocular levels obtained. However, the disappearance from the intraocular fluids was less rapid after the larger subconjunctival doses. No significant difference in this regard occurred with a 25-mg. as compared to a 12.5-mg. injection (fig. 3).

When subconjunctival injections are made in one eye and both eyes of a rabbit analyzed for the corticoid content, a slight increase is found in the intraocular fluids of the uninjected eye. The peak levels in the uninjected eye are slightly delayed in the aqueous humor of the uninjected eye, and even more delayed in the vitreous humor. The concentrations in the uninjected eye are decidedly lower than in the injected eye (fig. 4).

Retrobulbar administration produced low aqueous- and vitreous-humor concentration (fig. 2).

Intraocular penetration following intramuscular administration. It is evident from a study of Figure 1 that intramuscularly administered cortisone penetrated readily into the aqueous and somewhat less so into the vitreous humor. The peak levels are obtained in the aqueous humor within 24 hours, and are well maintained for 72 hours, although the number of instillations of cortisone per day were gradually reduced. However, the cortisone level in the aqueous humor diminished after two days of 100 mg. injected daily, so that within 24 hours after the last intramuscular injection, the concentrations approximated those of the normal aqueous humor.

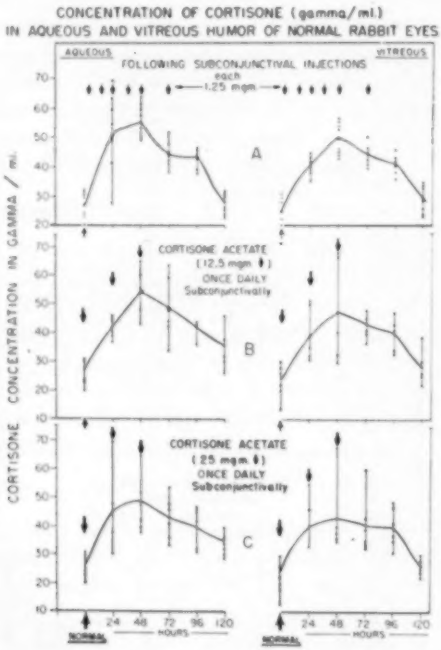

Fig. 3 (Leopold and Maylath). Comparison of cortisone injections of various strengths.

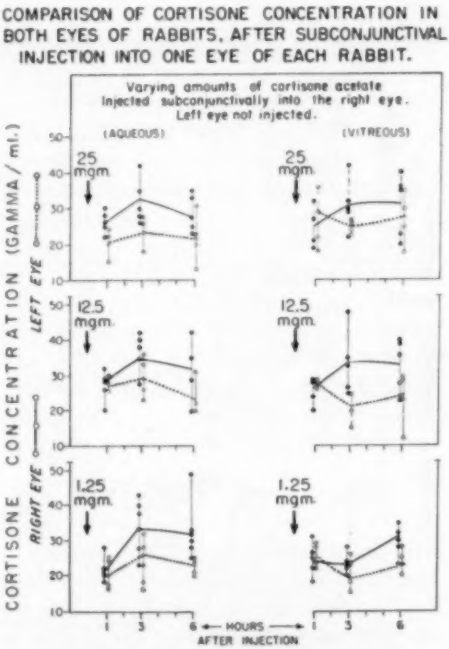

Fig. 4 (Leopold and Maylath). Concentrations of cortisone in injected and uninjected eyes.

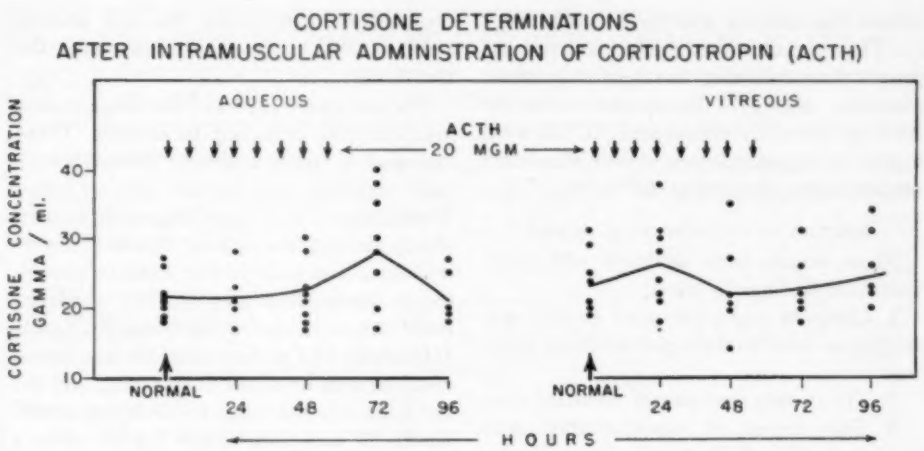

Fig. 5 (Leopold and Maylath). Corticoid concentrations after intramusclar injection of ACTH.

The concentrations of cortisone in the aqueous humor following intramuscular administration were equal to those obtained by subconjunctival injection in the dosage schedule used in this study.

The concentrations obtained in the vitreous humor reached their maximum after three days of therapy. These levels fell gradually thereafter and, within 24 hours after cessation of therapy, aproximated those obtained in the normal eye. The concentrations produced in the vitreous by intramuscular injection were not significantly greater than those obtained following subconjunctival injection in the dosage schedules used in this study.

The corticoid concentrations in the anterior segment began to rise only after two days of intramuscular corticotropin (ACTH) therapy. The rise in the vitreous

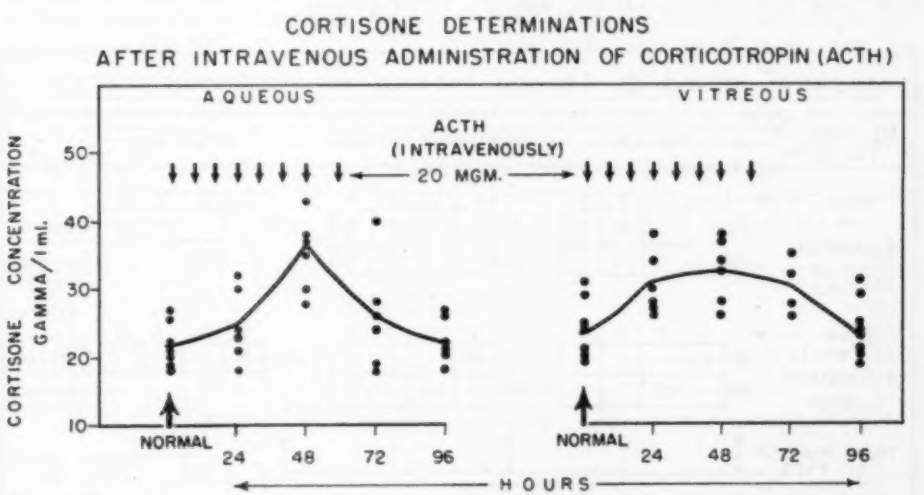

Fig. 6 (Leopold and Maylath). Corticoid concentrations after intravenous injection of ACTH.

humor was delayed another 24 hours (fig. 5). The concentrations reached did not approach those following two days of systemic cortisone therapy. Intraocular corticoid levels produced by intravenous ACTH were higher and appeared more rapidly than after intramuscular administration (fig. 6).

Results of experimental burns

These results were analyzed with three particular findings in mind:

1. Complete regeneration of corneal epithelium as detected through fluorescein staining.
2. The density and size of resultant scar.
3. The degree of vascularization was studied in the alkali burns alone. Vascularization did not occur in the thermal or acid burns of the treated or control eyes during the period of observation.

Thermal burns

It is suggested by the results in Figure 7 that the epithelial regeneration was retarded very slightly in the eyes treated topically, subconjunctivally, and systemically as compared to the control eyes. All but one control eye showed no staining by the third day; whereas, there were two eyes treated topically, four eyes subconjunctivally, and 18 eyes treated systemically that still showed small breaks in the epithelium until the fourth day.

The scars were evaluated according to area involved and according to density. Those classified as Grade-I severity showed a very faint scar that could be seen only by retroillumination. The iris markings could be seen clearly through the opacity. Grade-II severity was a faint cicatrix that could be seen by direct illumination and the iris markings could still be discerned fairly readily. Grade III consisted of a cicatrix which was dense, could be seen by direct illumination, and did not allow iris markings to be distinguished. Grade IV was dense which did not allow a visualization of iris and extended through the entire stroma.

It is evident from the study of Figure 7 that the density of the resultant cicatrix was much less in those eyes treated with cortisone topically, subconjunctivally, or systemically. Systemic therapy was not as efficacious as local therapy for thermal burns in this regard. Slightly less than 50 percent of the eyes treated systemically showed Grade-I cicatrices, while all of the eyes treated topically and subconjunctivally showed only Grade-I scars.

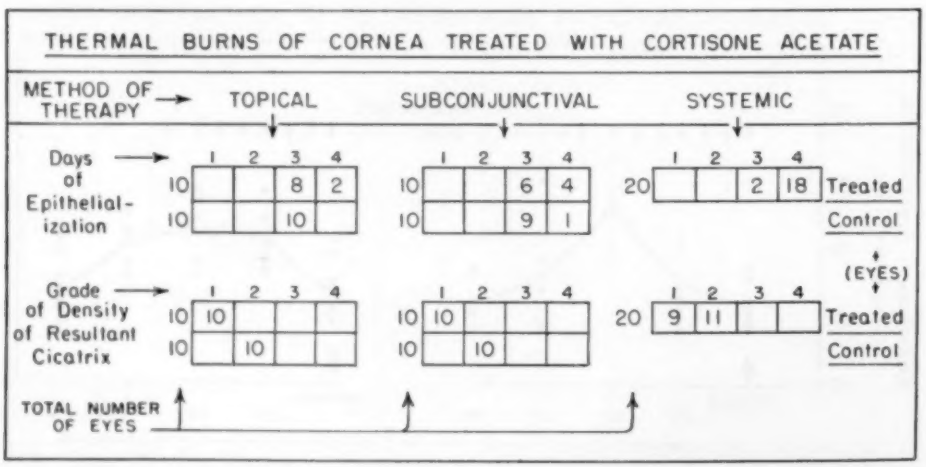

Fig. 7 (Leopold and Maylath). Results of cortisone therapy in thermal burns.

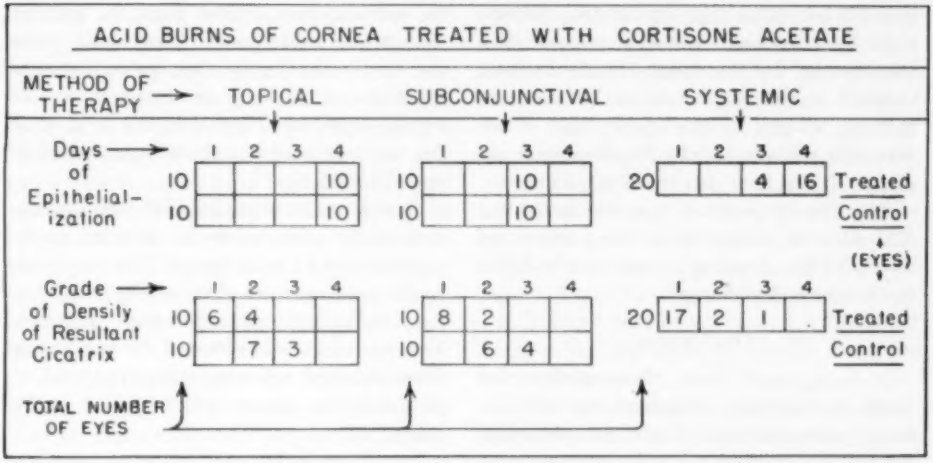

Fig. 8 (Leopold and Maylath). Results of cortisone therapy in acid burns.

Acid burns

Epithelial regeneration was retarded slightly only in those eyes which were treated systemically. This was a very slight difference. Fifty percent of the control eyes were regenerated in four days; 50 percent in three days, and 75 percent of the systemically treated eyes showed complete regeneration after only four days. All of the topically and subconjunctivally treated eyes were similar to their control eyes in the day of epithelial regeneration (fig. 8).

The density of the cicatrix was affected beneficially by cortisone whether the treatment was instituted topically or systemically. No superiority could be demonstrated for local versus systemic in the resultant cicatrix of acid burns.

Alkali burns

No significant difference could be demonstrated between the day of epithelial regeneration of the treated and control eyes following this severe alkali burn, with the

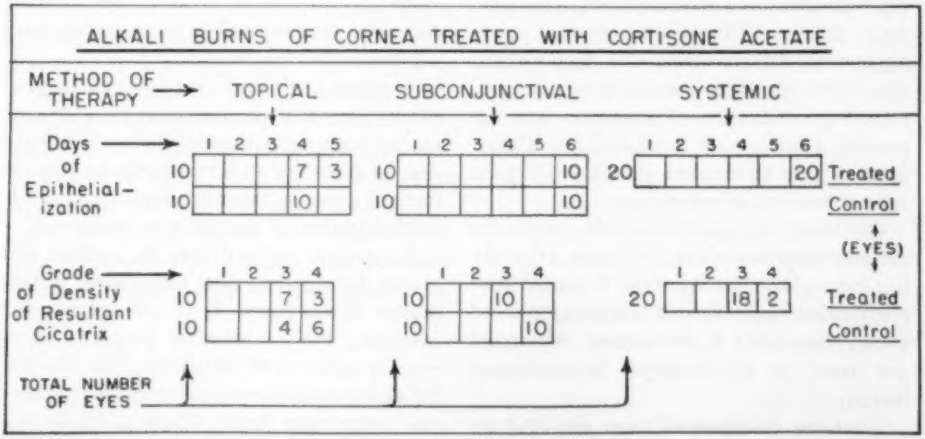

Fig. 9 (Leopold and Maylath). Results of cortisone therapy in alkali burns.

possible exception that topical drop therapy may have delayed epithelial regeneration slightly (fig. 9). The density of the resultant cicatrix was slightly reduced by cortisone therapy, so that by the seventh day of observation there was a slight difference in the degree of resultant density of the scars.

The day of onset of vascularization was delayed in the treated eyes. The number and length of the invading vessels were reduced in the eyes under therapy.

Discussion

It is apparent from these studies that cortisone, topically or systemically administered, penetrates readily into the intraocular fluids. This confirms analyses previously reported.[2]

Cortisone applied as drops of suspension, diluted 1:4 in 1:10,000 zephiran, penetrates readily through the normal rabbit cornea. Repeated applications over several days allow a maximum concentration to be reached which does not seem to be exceeded by continued treatment. Cortisone disappears rapidly from the anterior chamber as soon as therapy has been stopped. Within 24 hours, practically no detectable cortisone was present as determined by the method used in this study.

Subconjunctival cortisone allowed a more rapid penetration following application than drop therapy. The levels obtained were higher than those reached with drop instillation. Although slight increase in the vitreous-humor concentration of cortisone was detectable after use of drop therapy, significantly higher levels were obtained following subconjunctival administration.

Cortisone disappeared rapidly from the anterior chamber within 24 hours after the last subconjunctival injection. Repeated subconjunctival injections of cortisone allowed a maximum level to be reached, which did not seem to be exceeded by continued therapy.

Cortisone disappeared more slowly from the vitreous humor than from the anterior chamber, and disappeared more slowly from the intraocular fluids after subconjunctival injections of 12.5 mg. as compared to 1.25 mg. of cortisone. There does not seem to be any merit to subconjunctival injection of 25 mg. over 12.5 mg.

Systemic administration allowed concentrations of cortisone to be detected in the aqueous and vitreous humor. The maximum levels were reached after a longer interval than following subconjunctival injection. The vitreous levels obtained did not exceed those obtained following subconjunctival injection in the dosage schedule used in this study.

Intramuscularly administered corticotropin produces an increase in intraocular corticoid substances only after two days of therapy. Intravenous corticotropin decreases this latent period.

From these penetration studies it is apparent that all three methods should prove efficacious in the treatment of anterior segment lesions. Posterior segment lesions, particularly in the anterior portion of the posterior segment, should respond equally well to subconjunctival and systemic administration. However, the retina and choroid were not analyzed for their concentrations of cortisone and it is quite possible that the systemic route would allow greater concentration to be obtained in these tissues than subconjunctival injection.

For conditions involving the anterior portion of the posterior segment and the vitreous humor, subconjunctival injections would appear to allow one to obtain cortisone concentrations equal to those obtained by systemic administration, if not superior to them.

It must be realized that the method employed for analysis does not possess a high degree of accuracy. It is not specific for cortisone. For comparative purposes, however, it appears to be useful. The Porter-Silber reaction gives positive values for normal intraocular fluids. These normally oc-

curring corticoid substances are under investigation at the present time and the results will be reported in the future.

Two striking results were found in the thermal and acid burns following cortisone therapy. Regardless of the route of administration, all resulted in a definitely less dense scar formation on the treated side as compared to the untreated. The reduction in density and the extent of scar formation may be due to the inhibition of fibroblastic proliferation that has been observed repeatedly.[2-8]

Although no definite conclusions can be drawn from this experiment concerning the rate of regeneration of corneal epithelium, it appears that cortisone did not effect favorably the rate of regeneration of corneal epithelium following thermal, acid, or alkali burns. This slight retardation of corneal epithelial regeneration has been reported previously for corneal abrasions.[2, 3, 8]

It is interesting to note that not one of these eyes was the victim of secondary infection. This may mean simply that these animals all had an inherent resistance to the possible contaminants and secondary invaders, as it is known that cortisone has no direct antibacterial effect. As reported previously by Jones and Meyer,[9] Irvine,[10] Ashton, Cook and Langham,[11] and Lister and Greaves,[12] corneal vascularization after severe alkali burns was reduced by cortisone therapy.

There seems to be no advantage to systemic cortisone for the treatment of these lesions. All of the burns responded as well to topical or subconjunctival cortisone as to systemically administered cortisone. The results of this study would recommend a routine therapy of corneal burns with cortisone in an effort to reduce the subsequent scar formation.

Summary

1. Topically applied cortisone-acetate suspension penetrates readily into the anterior chamber of normal rabbit eyes. Continuous daily therapy allows a maximum level to be reached which is not exceeded by continued treatment. Cessation of therapy is followed by a rapid drop in the anterior-chamber concentrations of cortisone within the subsequent 24 hours.

Topical therapy with cortisone-acetate suspension allows detectable but not high levels of cortisone to be found in the vitreous humor. Cortisone disappears from the aqueous humor within 24 hours after cessation of therapy but not quite so rapidly from the vitreous humor.

2. Subconjunctivally administered cortisone produced high concentrations in the aqueous humor more rapidly than following drop therapy. Vitreous concentrations following this route of administration were higher than after topical administration. Within 24 hours after cessation of subconjunctival administration, the cortisone levels rapidly diminished in the anterior chamber and somewhat more slowly from the posterior chamber.

3. Systemically administered cortisone penetrated readily into the aqueous and vitreous humor. The levels obtained in the aqueous humor were higher than in the vitreous humor. The rate of penetration into the aqueous humor was slower following systemic administration than after subconjunctival administration. The rate of appearance of corticoid materials after intramuscular and intravenous corticotropin was slower and less in amount than after intramuscular cortisone.

4. All methods of therapy—topical, subconjunctival, and systemic—produced a less dense scar in thermal, acid, and alkali burns of the cornea as compared to untreated control eyes.

5. Cortisone therapy reduced the extent of corneal vascularization following severe alkali burns.

1930 Chestnut Street (3).

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DRILITOL IN EXTERNAL OCULAR DISEASES*

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Drilitol† is an isotonic aqueous solution prepared for the treatment of upper respiratory tract infections. It contains four active ingredients—two antibiotics (gramicidin and polymyxin-B sulfate), an antihistaminic (thenylpyramine hydrochloride), and a vasoconstrictor (paredrine hydrobromide). In addition, it contains a wetting agent (a quarternary ammonium-bromide compound) and a preservative (thimerosal). Consequently, the preparation is bacteriostatic, bacteriocidal, antiallergenic, and decongestant simultaneously. Though it has been extensively used in rhinitis, paranasal sinusitis, and nasopharyngitis, the analysis and dissertation herewith presented serves as the means of introducing Drilitol as an ophthalmic agent.

Since the ophthalmic literature contains but few references to gramicidin and the polymyxins (the antibiotics in Drilitol), a brief review of them should be of current value.

TYROTHRICIN, TYROCIDINE, GRAMICIDIN

Tyrothricin is a bacterial substance prepared from cultures of Bacillus brevis. It is antibiotic and specific against gram-positive bacteria. The two active principles derived from tyrothricin are gramicidin and tyrocidine. Of the two, gramicidin is the more potent antibiotic. Extensive research work has been done, in vitro and in vivo, with tyrothricin, tyrocidine, and gramicidin. Clinical application has been limited because of the toxicity which results when the agents are used intravenously. Some of the findings of this research are summarized here.

Studies on tyrothricin reveal it to be strongly hemolytic and toxic to formed elements of the blood and to tissue cells in general. Following intravenous administra-

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† Trademark of Smith, Kline, and French Laboratories.

tion, there occurs necrosis of the liver, spleen, and glomeruli of the kidneys.[1] Tyrothricin is not soluble in the body fluids nor is it dialyzable through the vessel walls. However, it is absorbed from the peritoneal cavity of laboratory animals with resultant damage to the liver, spleen, and kidney. (Gramicidin alone showed more extensive necrosis.) Though strongly "antigram positive," the ingestion of tyrothricin does not alter the gram-positive flora of the intestines.

Local applications of tyrothricin to the skin, mucous membranes, and infected pleura causes no toxic effect. Clinically, it has been used locally with success in acute otitis media, sinusitis, streptococcal dermatitis, chronic empyema, and following mastoid operations. Its application to fresh wounds and recent operative sites results in bleeding from the wound. This is due to its hemolytic activity; it is also leukolytic. Heath[2] used tyrothricin in the treatment of keratoconjunctivitis and pneumococcal conjunctivitis.

The optimum therapeutic concentration of tyrothricin has been determined at 500 μg. per cc.[3] No sensitivity to tyrothricin has been reported and it has proved effective in fantastically dilute concentrations.[4] Sodium chloride cannot be used as a diluent because it causes precipitation of the material. Extensive application of tyrothricin in rhinology, surgery, and dermatology revealed it to be a selective bacteriocidal agent against gram-positive cocci.[5]

Gramicidin, as has already been mentioned, is one of the active principles of tyrothricin and, as such, has a marked antibiotic activity specific against gram-positive bacteria and is inactive against gram-negative bacteria. Investigation, pursuant to gramicidin alone, indicates that 0.005 to 0.0025 mg. inhibits pneumococci; 0.005 to 0.01 mg. inhibits hemolytic streptococci; larger amounts prevent the growth of Streptococcus faecalis and viridans, of gonococci, staphylococci, diphtheria bacillus, and meningococcus. However, some strains of Staphylococcus aureus are most resistant.

Laboratory and clinical experimentation shows that hemolytic streptococcal empyema in rabbits is cured after the injection of gramicidin into the pleural cavity. In humans, superficial infections, due to staphylococcus and streptococcus, respond satisfactorily to local application of a gramicidin solution.[6] Suspensions of gramicidin, up to 0.5 percent, instilled in the conjunctival sacs of rabbits, caused no irritation. In Drilitol, the strength of gramicidin is 0.005 percent.

In contrast to tyrothricin and tyrocidine, gramicidin is not hemolytic and only slightly leukolytic. Moreover, the action of gramicidin is not inhibited in the presence of serum albumin. This is significant because the presence of albumin in exudates inhibits the efficacy of the antibiotics.

The polymyxins

The term "polymyxin" is applied to a group of related polypeptide antibiotics obtained from Bacillus polymyxa, a spore-forming rod occurring in soil. The polymyxins are notable for their high activity against gram-negative bacteria and their lack of such activity against gram-positive bacteria.

Four polymyxins have been isolated from different strains of Bacillus polymyxa and are designated A, B, C, and D. At its inception, the A was designated "aerosporin" and the D "polymyxin." (However, at present the B is referred to as "aerosporin.")

The polymyxins are stable under physiologic conditions of temperature and pH, both in aqueous solution and in powder. They are unstable under alkaline conditions.

In vitro, the polymyxin-sensitive gram-negative bacteria include Aerobactor, Brucella, Eberthella, Escherichia, Hemophilus, Klebsiella, Pasteurella, Pseudomonas, Salmonella, Sigella, and Vibrio.[7] Some species or strains are resistant occasionally; Proteus bacteria are generally resistant. Inclusion of blood or serum in the culture media does not affect the activity of the polymyxins. The polymyxins are rapidly bac-

teriocidal, the action appearing to be a lytic effect.

Bacterial sensitivity to the polymyxins is parallel in vitro and in vivo. A concentration of a dose is more effective than the same amount in divided dosage. Parenteral administration is more effective than oral. In mice, combatting K. pneumoniae, to obtain the same therapeutic response, oral administration required 64 times as much polymyxin as subcutaneous or intravenous administration.

Therapeutic blood levels are easily attained by parenteral administration of the polymyxins. Absorption from the gastro-intestinal tract also occurs. Cumulative serum concentrations are evident if doses are more frequent than at 12-hour intervals.

It is observed that the major toxic effects are those of renal damage. Minor disturbances are elevated temperatures, malaise, and nervous phenomena. These consist of paresthesias, hypethesias, dizziness, and weakness. In a group of children treated with polymyxin, intramuscularly, albuminuria was present in 50 percent and marked lethargy, irritability, and anorexia were noted in all.

Clinically, patients with diseases caused by polymyxin-sensitive bacteria responded well to parenteral treatment with the polymyxins. These diseases included peritonitis, pertussis, shigella enteritis, urinary tract infections and influenzal meningitis. However, the toxic reactions limit the use of the polymyxins to local therapy, preoperative preparation in abdominal surgery, and to those systemic infections whose gravity outweigh the temporary renal damage.

Granulating wounds infected with Pseudomonas aeruginosa yielded to topical treatment with polymyxin B. The strength of polymyxin-B sulfate in Drilitol is 500 units per cc.

No record of the development of any polymyxin- or gramicidin-fast bacteria exists nor is there any reported occurrence of sensitivity to either of these agents.

Newer developments in the usage of the antibiotics indicate a strong synergistic action between these drugs. An enhanced bactericidal effect is a present potential in the combination of gramicidin and polymyxin-B sulfate. An additional advantage of these antibiotics is their stability in solution—no deterioration in potency occurs over protracted periods. This contrasts favorably with the 48- to 72-hour stability of other antibiotics in solution.

Hydroxyamphetamine hydrobromide

Paredrine hydrobromide, whose chemical name is hydroxyamphetamine hydrobromide, is included in one-percent strength in Drilitol because of its decongestant effect on the nasal mucous membranes. In ophthalmology, paredrine has had long usage as a cycloplegic adjuvant and mydriatic. It is precisely this mydriatic effect which poses the disadvantage of its inclusion with antibiotics for ophthalmic use. However, the mild cycloplegic properties have not been significantly disturbing. The rapid blanching of the palpebral and bulbar conjunctivas is the principal function of paredrine in the solution.

Thenylpyramine hydrochloride

Thenylene and histadyl are the trade names of thenylpyramine hydrochloride, an antihistaminic drug. It is included in Drilitol for its value in alleviating allergic nasal conditions.[8] These allergic conditions are often indistinguishable from nonallergic types.

Antihistaminic agents have been used topically for several years in external ocular allergies. I reported the efficacy of the use of antistine,[9] histadyl,[10] and trimeton maleate[11] in many types of ocular allergy. Included were allergic conjunctivitis of hay fever and of other hypersensitive states; allergic dermatitis and urticaria of the lids; allergic blepharoconjunctivitis, episcleritis, and vernal catarrh.

Other reports[12] on histadyl include its successful use (to a lesser degree) in the

treatment of corneal erosion, phlyctenular keratoconjunctivitis, superficial punctate keratitis, chronic conjunctivitis, and blepharitis. Thenylpyramine is present in Drilitol in 0.2-percent strength. Other attributes of thenylpyramine, apart from the antihistaminic activity, are its mild decongestant and anesthetic properties.

Among the remaining ingredients of Drilitol are several that warrant consideration. Thimerosal (merthiolate) in concentration of 1:100,000 acts as a preservative and prevents mold invasion. This is one-half the strength used safely for many years in ophthalmic solutions. Eucalyptol is an essential oil which has been used in ophthalmology without irritation.

A wetting agent, a quaternary ammonium-bromide compound, is utilized in Drilitol to facilitate the miscibility of gramicidin, which is water insoluble. This chemical detergent is antiseptic and bactericidal in itself. It expedites the sterilization and absorption of drugs.[13] Therefore, it is used for preoperative cleansing of skin and conjunctiva, sterilization of instruments, and is a vehicle for drugs such as carbamylcholine chloride (carcholin). This wetting agent in Drilitol effects the reduction of the surface tension of the solution to a low of 35 dynes/cc. In this wise is enhanced the dispersion of the drugs to the nasal mucosa, for which use it is primarily adjusted.

Because of this low surface tension, the ocular use of the solution is associated with a mild congestion and burning sensation. Although the wetting agent has a number of advantages in ophthalmic use, it would be well to note that in the eye, where dispersion is easily attained, the surface tension might be increased.

Clinical application

From the foregoing, it is evident that the solution of a combination of chemotherapeutic agents, primarily intended for the nose, would be effectively applicable to ocular use. On this basis, 94 cases of various external ocular diseases were submitted to treatment with Drilitol solution. Included were cases of acute and chronic catarrhal conjunctivitis, squamous and ulcerative blepharoconjunctivitis, phlyctenular keratoconjunctivitis, dacryocystitis, allergic conjunctivitis, and vernal catarrh. All cases were typical in their categories.

Analysis of the cases revealed the following symptoms in order of their frequency; discharge (mucous or purulent), tearing, burning, foreign body or sandy sensation, itching, tired sensation, blurring of vision, and ocular soreness. In the main, all of these symptoms responded to treatment with Drilitol.

Undesirable manifestations, resulting from the ocular use of Drilitol, consisted of burning sensation, allergic reaction, dilation of the pupil, and one instance of sneezing. The burning varied from mild to severe but only one patient discontinued treatment on that account. Allergic reaction occurred in four cases—one developed urticaria; the others, contact dermatitis and conjunctivitis. In two of these cases, it was doubtful that the allergic reaction was due to Drilitol, inasmuch as there was concurrent exposure to other allergens.

Despite the fact that many light irises were slightly dilated as a result of the paredrine, only three or four of these patients were aware of it. This disadvantage of Drilitol solution could be obviated by replacing the paredrine with an ocular decongestant minus the mydriatic characteristics. A second improvement, the diminution of the burning sensation, could be effected by an elevation of the surface tension of the solution.

Table 1 is self-explanatory and shows that 76 of the 94 cases recovered completely.

Analysis of the 18 cases, in which Drilitol failed to effect improvement, reveals the majority as chronic types of conjunctivitis, resistant to all forms of therapy. The remaining unresponsive cases were amenable to treatment with other topical antibiotic or chemotherapeutic agents. The indication is

TABLE 1

SUMMARY OF CASES

	Improved	No Improvement	No. Cases
Acute conjunctivitis, catarrhal and purulent	30	3	33
Chronic conjunctivitis, catarrhal	29	9	38
Chronic conjunctivitis, angular	2	1	3
Blepharitis squamosa	6	1	7
Phlyctenular keratoconjunctivitis	2	0	2
Dacryostenosis with purulent discharge	2	3	5
Orbital conjunctivitis with integrated implant and purulent discharge	1	1	2
Allergic conjunctivitis with superimposed acute conjunctivitis and keratitis	1	0	1
Allergic conjunctivitis	2	0	2
Vernal catarrh, limbic variety	1	0	1
Total	76	18	94
Percentages	80.2	19.8	100

that the causative bacteria were resistant to gramicidin and polymyxin but not to other antibiotics.

In the 76 cases that reacted favorably to treatment, the rapidity of response varied, from 48 hours to five to 10 days. In all cases of recovery, the relief of symptoms and the disappearance of pathologic condition were impressive. The most striking results were obtained in the more acute conditions. The improvement was approximately 80 percent. Hence, it is evident that Drilitol is an effective addition to the ophthalmic armanentarium.

CONCLUSIONS

1. Presented are the results of 94 cases of external ocular diseases treated with Drilitol, a solution containing a combination of two antibiotics, an antihistaminic, and a decongestant.

2. In 80 percent treated with Drilitol, the therapeutic response was good or excellent. Included were various types of conjunctivitis, keratitis, and dacryocystitis.

3. The pharmacologic and clinical literature on tyrothricin, gramicidin, and polymycin-B sulfate are briefly reviewed.

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SUPRASELLAR MENINGIOMA*

Report of an atypical case

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Meningiomas account for 12 to 20 percent of all intracranial tumors, and about half of these are situated in the anterior half of the brain, usually at its base.[1] This is of particular concern to ophthalmologists for basal tumors sooner or later press on the optic nerves or chiasm causing visual defects.

Meningiomas are benign encapsulated tumors attached to some portion of the dura, falx, or tentorium. As a rule, their growth is slow. They vary in size and shape from small flat plaques a few millimeters in thickness to large irregular and nodular masses. If excised they do not recur, and even if removal is not complete the subsequent regrowth is quite gradual. X rays and radium do not have a favorable effect on these growths. Instead, scar tissue develops which makes subsequent surgical removal much more difficult.[2]

Meningiomas become more or less intimately attached to the neighboring dura and may even extend through it.[3] Their effect on adjacent bone varies considerably. In some, the process invades the haversian canaliculi causing hyperostosis. In others, the pressure of the tumor causes atrophy. And in still others, no known changes are apparent.[4] It is these latter cases which demand very thorough investigation, for one may be misled by the negative roentgenographic findings.

Whether or not bone changes occur depends to a considerable extent on the situation of the meningioma. This fact is of importance to ophthalmologists, because meningiomas arising from the region of the pituitary fossa are not likely to cause more than minimal bone defects.[3] And it is such tumors which may cause early visual symptoms.

Meningiomas have certain definitely favored sites of origin, each of which produces a more or less characteristic train of symptoms. Those primarily affecting the visual pathways have their origin near the optic nerve; from the lesser wing of the sphenoid; from the olfactory groove; and from the tuberculum sellae or sulcus chiasmatis.

Suprasellar meningiomas usually arise from the tuberculum sellae or the sulcus chiasmatis. Due to the close proximity of the optic chiasm and the prechiasmal portion of the optic nerves, either one or the other of these structures is soon compressed, even while the tumor is still small. Whether the chiasm or optic nerve is first affected depends on the situation and direction of growth of the meningioma, as well as the situation of the chiasm.

In typical cases the pressure is chiefly exerted on the chiasm, which is forced upward and backward, causing a disturbance of function of, and damage to, the affected visual fibers. This tends to produce bitemporal field defects and the picture of primary optic atrophy.

Cushing[5] first called attention to the characteristic features in these cases. Noting that meningiomas are largely confined to adults, and that those arising from the sella turcica usually cause little or no reactive hyperostosis, he conceived the idea that these features could be grouped together to form a syndrome characteristic of suprasellar meningiomas.

He described what he called the "chiasmal syndrome" as consisting of primary optic atrophy with bitemporal field defects, which are not necessarily symmetrical, in adult pa-

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tients showing an essentially normal sella turcica. He pointed out that the syndrome is not always consistent, and is subject to considerable variations in all of its features.

Closer scrutiny of the nature of the process readily explains these variations. For instance, meningiomas sometimes occur in young individuals. Also, while meningiomas arising in this region are not apt to cause gross bone defects, modern and more refined roentgenographic technique has revealed that minimal changes occur more frequently than was formerly thought. But, of greater importance from the ophthalmologist's point of view are the variations in the visual field defects, and, to a lesser extent, those concerning the degree of resultant optic atrophy.

Schlezinger, Alpers, and Weiss,[6] show in their study of the anatomic relationships that suprasellar meningiomas may primarily involve the prechiasmal portions of the visual pathway rather than the chiasm itself. This causes quite a different perimetric picture. For, in prechiasmal cases the visual function of the two eyes is attacked successively rather than simultaneously. As a consequence, vision in one eye may be seriously impaired while the other eye remains normal or nearly so. Then after a variable period, extending from weeks to months or more, the second eye becomes affected.

The field defects in these cases are usually bizarre, and consist of a combination of central and peripheral involvement. One of the characteristics is the rapid development of central scotomas. Schlezinger and his associates expressed the belief that the relatively early appearance of central scotomatous field defects has not been sufficiently emphasized, and that the presence of an incomplete central scotoma is significant in the early diagnosis. In their cases, a later characteristic was a peripheral indentation which sooner or later merged with the central scotoma to produce an expanding sector defect.

It is difficult to understand why meningiomas cause a central field defect so regularly, whereas, adenomas in a similar location so rarely do. Mooney and McConnell,[7] in commenting on this incongruity, pointed out that a central scotoma is particularly likely to occur when the optic nerve is elevated or depressed in the region of the optic foramen. They referred to the possible mechanisms involved, such as pressure of the nerve against the margin of the foramen, and impaired nutrition from pressure on or kinking of the ophthalmic artery.

As in the other features of the syndrome, the appearance of the optic disc may be quite variable and is not always of assistance in the diagnosis of early cases. Since atrophy of the disc is dependent on injury of the fibers of the visual pathway, its degree varies with the amount of damage. Moreover, atrophy may not appear for some time after the field defect has become well established. Hence, the disc may appear normal or go through all the gradations of pallor to complete atrophy. And, although the ophthalmoscopic picture is characteristically that of primary optic atrophy, occasionally the disc margins appear blurred.

The ophthalmologic signs of suprasellar meningiomas are particularly important because of the general absence of other significant clinical features, at least in early cases. Physical and neurologic examinations are usually fruitless, and there are no characteristic subjective symptoms. It is true that pain or headaches are frequent and that visual hallucinations are not uncommon. But their presence is not consistent and hence cannot be depended on.

The inconsistencies of the chiasmal syndrome are so apparent that it is perhaps not strange that so many cases reported in the neurologic literature have escaped diagnosis until the tumor has become far advanced. If this occurs, not only is vision permanently destroyed but life is at stake. For as the tumor enlarges, it causes additional damage and produces a more complicated picture.

The pressure tends to cause absorption of the sella and the resultant signs of dispituitarism. As in other tumors in this region,

pressure or extension not infrequently cause paralysis of the extraocular muscles, papilledema, and impairment of the olfactory sense.

Other changes which may follow are distortion of the third ventricle and secondary hypothalamic symptoms, and ultimately, occlusion of the foramen of Monro, causing internal hydrocephalus.

Fortunately, the ophthalmologic symptoms are produced while the lesion is still small and easily removable, and usually before permanent damage has been done. However, in atypical cases this very fact tends to make the diagnosis more difficult because of the lack of other guiding symptoms.

Although the presence of a suprasellar meningioma cannot be positively determined by the field changes alone, one should gravely suspect such a lesion in cases of bitemporal field defects in adults who show no other symptoms, and in whom roentgenographic changes are minimal or absent. Moreover, a unilateral visual defect of unexplained origin should be viewed with suspicion for it is characteristic of prechiasmal cases. As these tumors grow very slowly, some time may elapse before the second eye becomes affected, and meantime irreparable damage may occur. Hence, every effort should be made to determine the true nature of the condition as early as possible. The slow growth of meningiomas does not preclude a sudden loss of vision, for a central scotoma may suddenly appear in an eye with a slowly contracting peripheral field.

In such cases of unexplained visual defects, further studies are indicated. Examination of the spinal fluid may be diagnostically significant if it shows a positive serologic reaction, an abnormal colloidal gold curve, or an elevated total protein content.[6] However, this does not offer positive proof, and since spinal puncture is not without danger in such conditions, the end may not always justify the means.

On the other hand, angiography and pneumoencephalography are so revealing that either one of these procedures is indicated in suspected cases showing negative plain roentgenographic findings. One advantage of angiography is that it not only demonstrates displacement of the arteries, but the meningioma itself may take up the radiopaque substance, outlining itself by the so-called "blush."[8] It should be noted that neither of these procedures is infallible, particularly early when the tumor is very small. In the event that all examinations are negative in a suspected case one may be justified in undertaking an intracranial exploration.

The following case is reported because of its atypical features, and to point out the value of angiographic studies even though long delayed.

Case report

History. A woman, aged 62 years, was examined October 15, 1947. The family history was noncontributory, and except for bilateral deafness which had followed an attack of typhoid fever many years ago, the past history was inconsequential. I had examined the patient June 17, 1942. At that time her only complaint was blurred near vision, and her eyes appeared normal except for the presence of myopic astigmatism and presbyopia for which glasses were prescribed. Vision was 20/30 in each eye, corrected to 20/20 and J1.

The present difficulty had developed three weeks earlier when vision in the left eye suddenly failed. She insisted that the onset was abrupt, and that within three or four days the sight was completely lost. She volunteered the information that for a day or so before vision was affected, the eye felt painful and tender, particularly on moving it. But these symptoms had gradually subsided, and now her only sensation was deep-seated orbital discomfort as she shifted her gaze. She denied the presence of any other symptoms. She had not had a headache in years and had not noted the occurrence of any visual hallucinations.

Examination. Aside from the presence of

myopic astigmatism and presbyopia, the right eye seemed to be normal in appearance and function. Vision was 20/30, corrected to 20/20 and J1. The peripheral and central fields were normal to white and colors.

The left eye appeared normal externally. It was not tender, the corneal sensitivity was not affected and its motility was not impaired. However, movement did cause definite orbital discomfort.

Vision in this eye was reduced to hand motion. The form field appeared to be grossly normal. The pupil was slightly larger than its fellow and was almost immobile to direct light, but reacted promptly consensually and to near presentation. The optic disc showed moderate temporal pallor. The lamina cribrosa was distinct and the margins clear-cut. The intraocular pressure measured 19 mm. Hg. (Schiøtz) and the exophthalmometer reading was 14 mm. (Hertel) in each eye.

A tentative diagnosis of retrobulbar neuritis was made and the patient referred to her physicians for investigation. They found a tendency to mild hypertension, but otherwise the general systemic examination gave entirely negative results. Except for the disability of the left eye, there was no evident impairment in the function of the cranial nerves, nor in that of the muscles, the reflexes, sensation, or coördination.

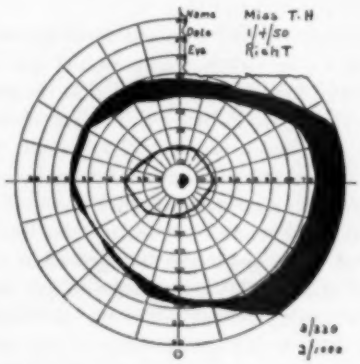

Fig. 1 (Joy). Visual field of the right eye, January 4, 1950, showing temporal contraction of about 15 degrees and an hemianopic central scotoma.

Otolaryngologic examination revealed bilateral otosclerosis but no other abnormalities. All roentgenograms, including those of the skull and orbit, failed to disclose any significant changes. The Wassermann reaction was negative. On one occasion the urine showed a trace of albumen, but on subsequent tests it was found to be normal.

Course. The patient did not return as directed and was not seen again until September 28, 1949, almost two years later. It was then apparent that the diagnosis of retrobulbar neuritis was hardly tenable. For, instead of improvement, vision in the left eye had completely failed and atrophy of the disc was complete. But the right eye was still normal in appearance and function, showing unimpaired vision and fields. The patient had become reconciled to the loss of sight in one eye and since there was no defect in its fellow, she would not consent to further studies.

She was convinced that further investigation was necessary when three months later she noted blurring vision of the right eye. This was two years and three months after involvement of the left eye. She appeared for examination January 4, 1950, stating that the sight in the right eye began to fail three days previously. Vision in this eye was 20/40 minus, not improved with glasses. The eye was normal in appearance, but perimetric examination revealed a temporal field contraction of about 15 degrees and an irregular hemianopic type of central scotoma (fig. 1). The left eye was unchanged.

It was now apparent that the cause of the trouble must be a lesion, probably a slowly growing tumor, in the region of the chiasm. This seemed to call for angiographic studies. But first, it was decided to repeat the previous examinations. Again, they disclosed no significant abnormalities.

The roentgenograms were studied with particular care. They showed the sella turcica

to be normal and no destruction of the clinoid processes. The left petrous pyramid had a large pneumatic cell near its tip, but otherwise the petrous pyramids were similar and normal. There was no bone destruction at the base of the skull.

Reëxamination with spot films in an effort to determine the presence of erosion failed to demonstrate any evidence of this condition. The optic foramina were normal in diameter, and the superior orbital fissures were demonstrated as normal on the two sides. The pineal body showed no evidence of shift.

The patient lived in a somewhat distant community, and this, together with a tendency to procrastinate, caused a delay of two months before angiographic examination could be undertaken. Meantime, vision in the right eye had dropped to 4/200, the right pupil had become sluggish in reaction to light, and the corresponding optic disc evinced a suspicion of beginning pallor. Also, the peripheral field showed a further temporal contraction of 15 degrees and the central defect had developed into a pericentral scotoma (fig. 2).

The angiographic examination was done by Dr. Arthur Ecker, on March 16th. It revealed good filling of the branches of the anterior and middle cerebral arteries, and the indirect vertebral angiogram showed filling of the basillar artery and a portion of the posterior cerebral artery. There was a definite elevation of the first portion of the anterior cerebral artery on the right, with an increase in density beneath this area suggesting the intrinsic circulation of a meningioma. There was no displacement of the basilar or internal carotid arteries to suggest the possibility of a craniopharyngioma. A diagnosis of meningioma was made (figs. 3 and 4).

This was confirmed on March 20th, when Dr. Ecker exposed a suprasellar meningioma through a right frontotemporal bone flap. The olfactory nerve, the optic nerve, and the tumor lying under the optic nerve could be seen very clearly. The tumor was pushing the optic nerve laterally and upward and somewhat forward. About four sq. cm. of the mass, which biopsy showed to be a meningioma, was removed with pituitary forceps.

The patient withstood the operation well and the postoperative course was uneventful. Vision improved rapidly, and five days after the operation she could read headlines with the right eye, and the temporal field was somewhat less contracted. She was discharged from the hospital March 31st. The final diagnosis was meningioma of the tuberculum sellae.

There was steadily progressive improvement in the succeeding months. By May 9th, less than two months after operation, corrected vision had improved to 20/20, the central scotoma had disappeared, and the peripheral fields showed little temporal contraction.

Maximum improvement was reached six months after operation and this has been maintained up to the present time. Vision in the right eye is now 20/30, corrected to 20/20 and J1. The visual field is normal. The optic disc shows questionable pallor. Otherwise, the eye shows no defects. The left eye remains unchanged. The patient feels

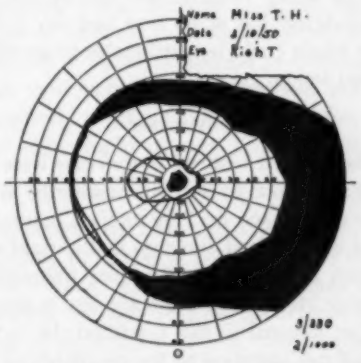

Fig. 2 (Joy). Visual field of the right eye two months later, showing further temporal contraction and enlargement of the central scotoma.

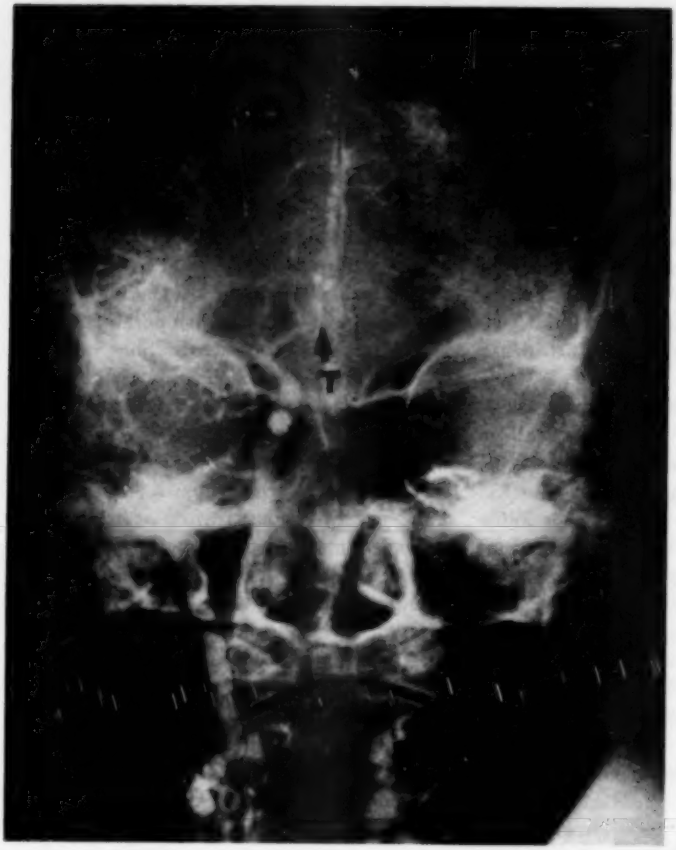

Fig. 3 (Joy). Anteroposterior arteriogram after injection of right carotid artery. The anterior cerebral artery, after its origin, ascends as it approaches the midline. ↑ indicates displacement of anterior cerebral artery by tumor. T indicates site of tumor.

well in every respect, and except for the visual defect has at no time had any symptoms which could be attributable to an intracranial lesion.

Comment

The atypical features in this case were the close simulation to retrobulbar neuritis in the early course, and the long interval before the onset of impaired function in the right eye. Sudden unilateral visual failure followed by atrophic changes of the optic disc is not, of course, unusual in this condition. In fact, it should call attention to the possibility of the presence of a suprasellar meningioma.

In this case, however, there was also pain and tenderness, giving the complete picture of retrobulbar inflammation. This is most difficult to explain unless we can assume that it was coincidental, or that the sheaths of the optic nerve were affected as a secondary result of intracranial pressure on the optic nerve. I am convinced that it was not imaginary, for the information was volunteered without suggestion, and residual evidence remained when I first examined the patient.

The second feature is less unusual, but it has not received the attention in the literature that it deserves. For suprasellar meningiomas may initially involve the optic nerve rather than the chiasm. Such was true in this case, in which the predominant characteristics were the successive involvement of the two

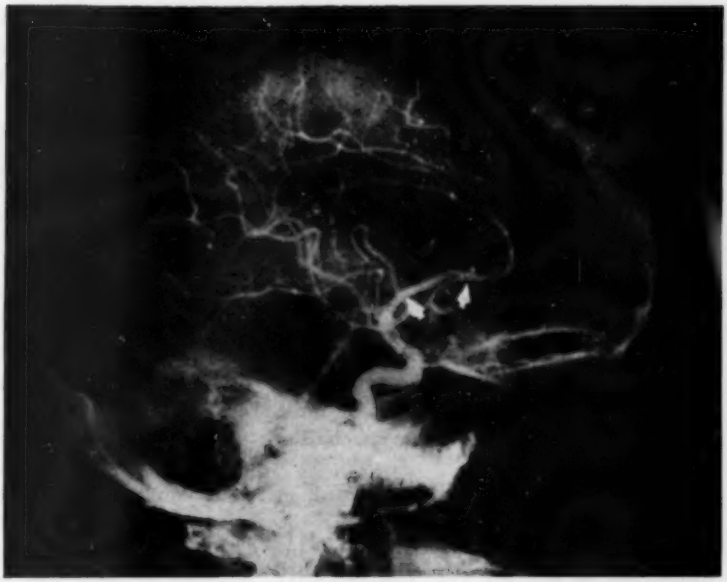

Fig. 4 (Joy). Lateral arteriogram, showing marked upward and backward displacement of right anterior cerebral artery. Suggestion of intrinsic circulation in tumor. ↑ indicates displacement of anterior cerebral artery by tumor. ▼ indicates site of tumor.

eyes, and the rapid development of central scotomas.

The fact that over two years elapsed before the function of the right eye was affected demonstrates how slowly meningiomas may grow. It also illustrates the fallacy of depending on the appearance of field defects in the second eye in the early diagnosis of these lesions.

The somewhat rapid progress of the field defects in the right eye might suggest a faster growing tumor than was apparent by the long delay before their original appearance. However, consideration of the anatomic relationships shows that this is not necessarily so. For, after a tumor has reached a certain size in this region, very little further growth is required to cause progressively increasing pressure on the neighboring structures.

In this case, early angiographic studies would undoubtedly have shown the true nature of the condition. It is likely that it would then have been possible to remove the tumor *in toto*, and with a good chance of restoration of vision in the left eye. But the patient disappeared for almost two years after her first visit, and angiography was delayed until the right eye was seriously affected. Fortunately, she now has normal function in an eye which was fast approaching blindness. And it is possible that she will retain it without submitting to a second operation.

This case illustrates the importance of complete investigation in the presence of unexplained visual defects. It shows why an apparent unilateral retrobulbar neuritis with recovery of vision should not be dismissed as such until intracranial involvement has been completely ruled out. This may require cerebral angiography or some similar procedure.

Summary and Conclusions

Meningiomas represent one of the more frequent and hopeful types of brain tumor. If discovered early they can ordinarily be removed without fear of recurrence. And even if extirpation is not complete subsequent regrowth is usually quite slow.

Suprasellar meningiomas are of particular concern to the ophthalmologist because the

first signs, and often until very late, the only signs are in his domain. As a rule, subjective symptoms are not constant, helpful neurologic findings are rarely present, and plain skull films show little or no bone changes. In typical cases, pressure on the chiasm causes early bitemporal field defects and the resultant picture of primary optic atrophy. However, if the prechiasmal portions of the visual pathways are primarily involved, the train of symptoms is quite different. For, the function of one eye may be impaired for a considerable time before the other eye is affected. And, since the rapid development of central scotomas is characteristic of these cases, the condition may simulate retrobulbar neuritis.

Such was true in the case presented, in which the outstanding features were the sudden onset of an apparent retrobulbar inflammation in the left eye, followed over two years later by temporal and central field defects in the right eye. No other significant signs or symptoms were ever present, and plain skull films failed to disclose any significant abnormalities.

The persistence of the visual loss in the left eye indicated that the tentative diagnosis of retrobulbar neuritis was not correct. Unfortunately, circumstances caused delay of angiographic studies until the right eye was seriously affected. By then it seemed probable that we were dealing with a slowly growing tumor in the region of the chiasm.

Angiography suggested the presence of a meningioma, which at operation proved to be suprasellar and compressing the right optic nerve. Its partial removal was followed by rapid restoration of normal function in the right eye.

This case exemplifies the variations which may occur in the train of symptoms associated with suprasellar meningiomas, and calls attention to consideration of primary prechiasmal involvement as an entity. It shows why the possibility of such a lesion should be suspected in the presence of an apparent retrobulbar neuritis, even if unilateral, if vision is not restored.

The long interval before the right eye was affected demonstrates how slowly meningiomas may grow, and at the same time illustrates the fallacy of depending on the appearance of field defects in the second eye in the early diagnosis of these lesions.

Finally, this case shows the importance of angiography as a diagnostic procedure in a condition which is notable for its lack of positive findings.

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CHORIOPATHY*

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An unusual case of choroid disease is presented as an etiologic and therapeutic problem.

R. J., a 40-year-old physician, was first seen March 31, 1944, because of photophobia, flickering lights, floaters, and severe frontal headaches. The head pains had been present for 20 years, but of late they had been more intense, especially on the left side. During the past five years he had several attacks of conjunctivitis and marginal corneal ulcers.

Family history. Mother living, aged 74 years. Father died at the age of 68 years from a heart attack.

Personal history. When aged 12 years, he was in bed several weeks with rheumatic fever without heart involvement. He has had measles, mumps, whooping cough, and chickenpox. His tonsils were removed 10 years ago because of a few attacks of sore throat and some indefinite joint pains. A fistula *in ano* promptly healed after operation. In 1937, he passed a kidney stone. He never used alcohol or tobacco excessively, and not at all in the past three years.

He is married and has had two children, one a daughter living and well; the other, a son, died of lymphosarcoma when 17 years of age.

General physical examination. Weight, 265 pounds. No enlarged lymph or other nodes anywhere in the body. The heart, lungs, abdomen, and teeth were normal. The blood pressure was 142/78 mm. Hg.

On repeated blood examinations, the red blood cells ranged from 3,700,000 to 4,890,000. The white cells remained 7,500 with a very slight variation in the differential counts:

Polynuclear neutrophils 62
Mononuclear neutrophils 1
Small lymphocytes 30
Large lymphocytes 3
Eosinophils .....1 to 4 to 6
Platelets .....250,000 cu. mm.
Hemoglobin .....77 percent to 106 percent
NPN 29.8
Urea N. 17.9
Uric acid 6.0
Serum chlorides .....550 mg. per 100 ml.
Calcium 11.4
Phosphorus 4.4
Cholesterol .....170 to 173
Blood sugar from 87 mg. percent to 117 mg. percent
Bleeding time, two minutes
Coagulation time, four minutes.

In a urine specimen of 1,115 cc. in 24 hours there were no albumin, no sugar, a few fine granular casts, and an occasional epithelial cell. At one time a positive reaction for porphobilinogen was obtained with a modified Ehrlich's reagent. This was not present in any of the several subsequent specimens.

Basal metabolic rate, —1.
Mantoux, +1 reaction
Rumpel-Leede test for capillary fragility, negative

After an intradermal susceptibility test of 0.1 cc. of histamine diphosphate, a 1.5 cm. wheal developed and at the end of 20 minutes was 2.8 cm.

The X-ray examination of the skull showed that the frontal bones were unusually dense and the inner table thick. The sella turcica was not enlarged or eroded, and the anterior and posterior clinoid processes were not remarkable. The pineal gland was calcified and in the anteroposterior view was directly in the midline. There were no abnormal calcifications within the skull. The right frontal sinus was very poorly developed, probably a supraorbital ethmoid. The remaining sinuses were well developed and clear throughout. The sphenoid was

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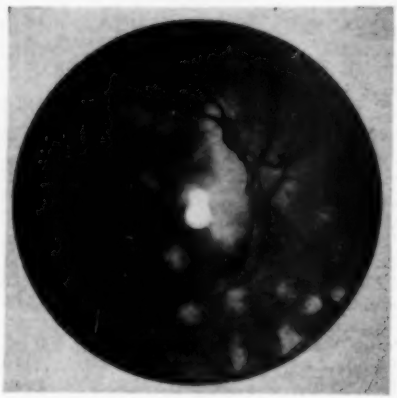

Fig. 1 (Bedell). Choriopathy, right eye.

clearly demonstrated and showed no evidence of pathologic changes.

Fundus changes

The chronologic detailed changes in the fundi are considered first in the right and then in the left eye.

Right eye (fig. 1)

March 31, 1944. Vision 6/15, with correction 6/5. The pupil was 3.0 mm. regular and active to light and accommodation. The media were clear with the exception of some very small, dustlike opacities and a few fine threads in the vitreous. The disc was clearly outlined with no central excavation. The retinal vessels were normal in distribution, size, and caliber. Beneath them were six soft, pale pink, round or oval flat areas* devoid of pigment close to the lower margin of the disc. The borders were not clearly defined. Between the macula and the disc there was a slight paling of the background. The choroidal vessels were not visible in any portion of the fundus.

May 3, 1947. Vision with correction, 6/7.5. There was a very marked increase in the number and the visibility of the areas not only in the region where they were first noted, but also in a wider zone about the disc especially in the lower nasal half of the fundus, where some were very irregular in form and a few, long broad streaks. The retinal vessels passed over all of them.

March 17, 1949. Vision 6/20, with correction 6/7.5. There were more spots in the lower nasal quadrant. Most of them remained soft, pale pink, with an occasional sharper edged paler one. Some large, confluent masses were irregular in form.

In the macular region there were many small dots remotely suggestive of drusen in size and distribution but lacking the typical color and reflections. To the outer side of the fovea there were a few pinpoint retinal hemorrhages. The temporal side of the disc was paler, and one spot impinged upon the lower temporal margin. There was practically no further alteration in the inferior portion of the fundus and no pigmentation.

May 26, 1949. The most striking change was the great increase in the number of spots, which were plainly visible and widely distributed even beyond the temporal side of the macula. Near the fovea there were a few minute, superficial retinal hemorrhages (fig. 2).

November 3, 1949. The only evident change was an increase in the number of the hemorrhages, which formed a thin, narrow arc near the inferior temporal side of the macula.

September 14, 1950. The disc was paler and a fresh infiltration overlapped the superior temporal margin. The macula was more granular. The hemorrhages had disappeared.

April 5, 1951. Vision 6/20, with correction 6/20. The disc remained well defined. The temporal half was paler with two overlying, cloudy yellow-pink areas, one above and the other below. In the macular region there were several fine, granular, yellow dots and a silvery, weblike reflex. A recent detachment of the retina in the lower half of

* In this report, "area" and "spot" are used synonymously to denote the lesion.

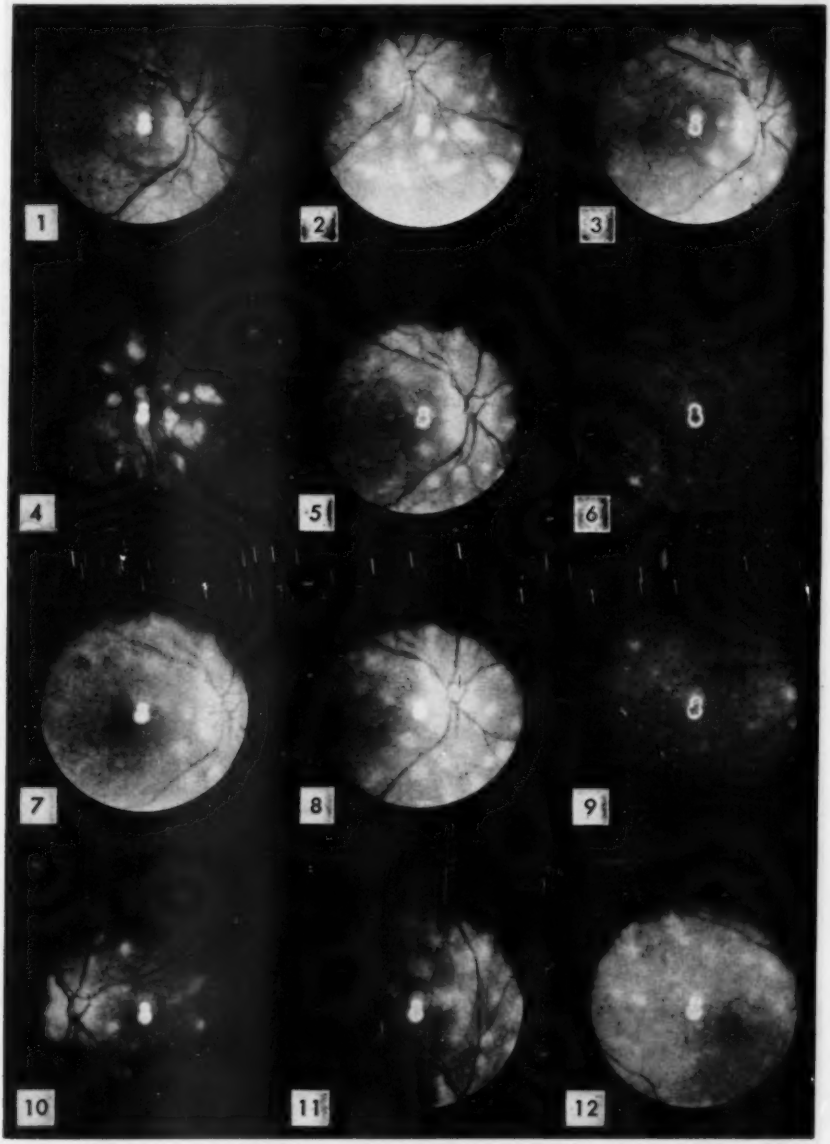

Fig. 2 (Bedell). Choriopathy, right eye. (1) March 31, 1944, central. (2) May 3, 1947, inferior. (3) May 3, 1947, central. (4) March 17, 1949, inferior. (5) March 17, 1949, central. (6) May 26, 1949, macular. (7) November 3, 1949, central. (8) September 14, 1950, central. (9) April 5, 1951, macular. (10) April 5, 1951, nasal. (11) April 5, 1951, superior. (12) April 5, 1951, macular.

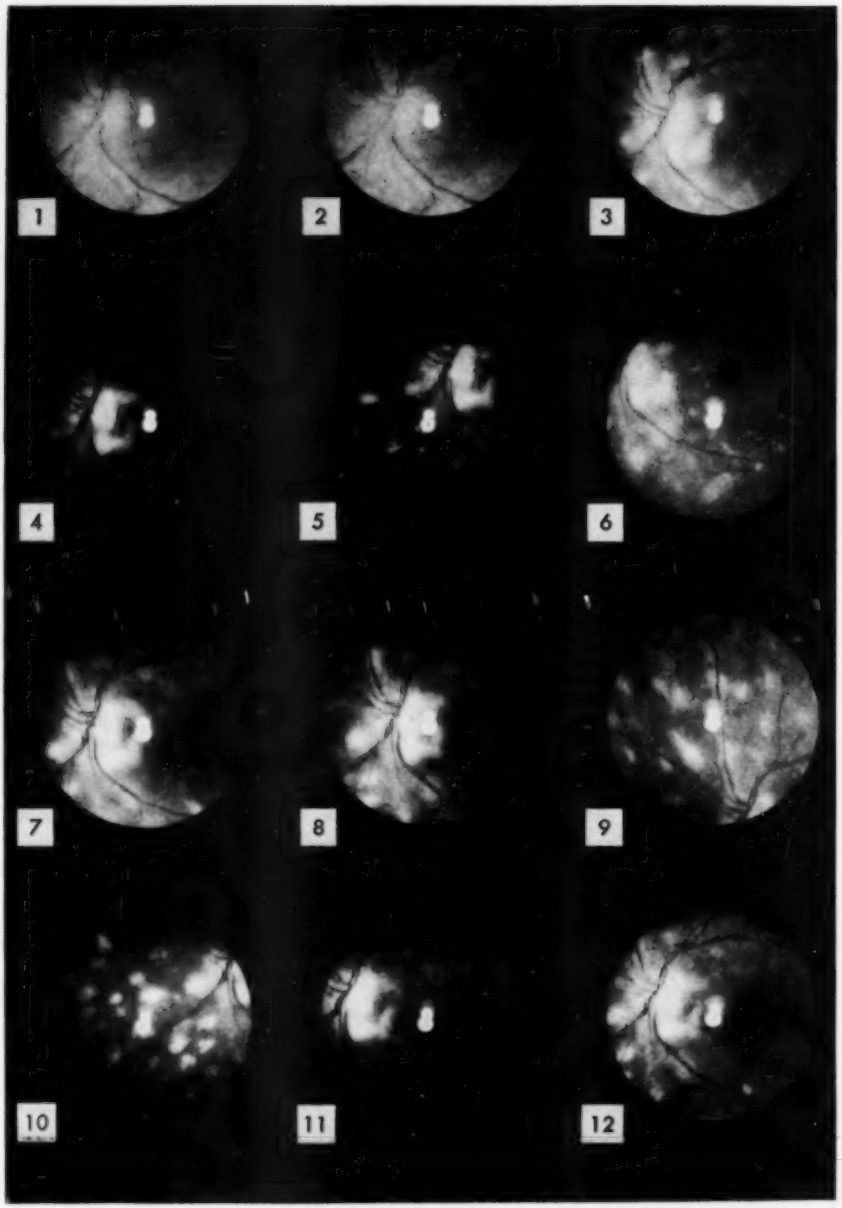

Fig. 3 (Bedell). Choriopathy, left eye. (1) March 31, 1944, central. (2) March 31, 1944, stereoscopic. (3) May 3, 1947, central. (4) September 18, 1947, central. (5) March 17, 1949, inferior. (6) March 17, 1949, central. (7) May 26, 1949, central. (8) May 26, 1949, stereoscopic. (9) April 5, 1951, superior. (10) April 5, 1951, nasal. (11) April 5, 1951, central. (12) April 5, 1951, stereoscopic.

the fundus bulged far forward and the spots were clearly seen to be confined to the choroid. The inferior temporal artery was slightly reduced in size. Throughout the fundus, the areas were more numerous, more varied in shape, larger and devoid of pigmentation.

LEFT EYE (fig. 3)

March 31, 1944. Vision 6/60, with correction 6/15. The pupil was 3.0 mm., regular, active to light and accommodation. The media were clear, with the exception of some very small, dustlike opacities and a few fine threads in the vitreous. The disc was well outlined, with a pale temporal half and no central excavation. The retinal vessels were of normal size and distribution. There were three soft, badly defined, pale, yellowish spots in the choroid below the disc, many less distinct ones on the nasal side and a few, almost invisible, above the pink macula.

May 3, 1947. The disc was slightly pale with an indefinite border, particularly in the lower nasal portion. The veins and arteries were of normal size and distribution. Infringing upon the lower temporal margin was a soft, yellow-white area about one-fifth disc diameter in size. Slightly separated from it was a larger, J-shaped mass with an almost vertical limb. Beneath the retinal vessels were many rounded, flat, nonpigmented spots. Some were paler than others. The macula was of normal color and surface configuration and above it the spots were especially thin, almost nebulous, clouds.

September 18, 1947. Vision 6/30, with corrective 6/7.5. In addition to the spots previously described there were granular, super-

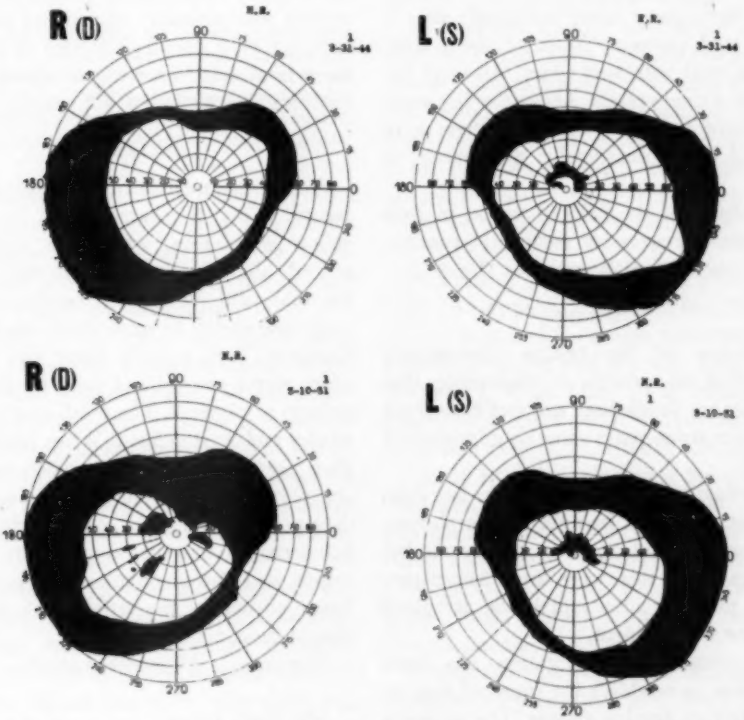

Fig. 4 (Bedell). Choriopathy, fields. Changes in the field, 1/330 white target, artificial illumination. Progressive contraction with several small scotomas.

ficial, globular red retinal hemorrhages in an almost horizontal line close to the inferior border of the macular region. The choroidal areas were larger and less regularly curved in outline.

March 17, 1949. The only change in the disc was a more marked pallor of the temporal side. There were many more spots to the nasal side and in the lower nasal quadrant.

May 26, 1949. The spots adjacent to the disc and most of those in the rest of the fundus were paler. The macular region was granular, with small, light-colored, irregularly arranged dots and one small superficial retinal hemorrhage inferior to the fovea.

April 5, 1951. A pale spot was close to the upper, temporal margin of the disc, and a larger, much paler one was over the inferior edge.

Scattered throughout the entire visible fundus were many spots completely devoid of migrated pigment. Most of them were round or oval with soft edges. All were beneath the retinal vessels. Above the disc some were almost as long as the disc, others were V-shaped and occasionally arrowhead in form.

The study of the field of vision in each eye showed progressive peripheral contraction with several small scotomas (fig. 4).

Summary

A review of the fundus photographs proves that the process is continuing, that new areas are developing, and that even those present for seven years show no migration of pigment.

An inflammation of the choroid has been ruled out, not only because of the absence of pigmentation in or about the lesions but also because if this were an inflammatory process the choroid would be be atrophied and white scars evident.

An exhaustive investigation has been undertaken to establish the cause of this remarkably rare fundus disease. The serologic reactions for syphilis and undulent fever were negative. There was no evidence of local or general tuberculosis. The spots are not miliary tubercles. Tubercles are ephemeral and never persist for years. Repeated blood examinations have failed to show any abnormality with the possible exception of a slight variation in the eosinophils 1 to 4 to 6 percent.

There has been no kidney dysfunction. Porphyrinuria was eliminated because it was present only in one urine specimen and there are no clinical suggestions of the condition. An allergic reaction to histamine was noted on only one occasion, and collagenous degeneration was not demonstrable. Disease of the accessory nasal sinuses was excluded by X-ray and local examination. There were neither neurologic symptoms nor physical signs of any intracranial lesion. Periarteritis nodosa was seriously considered but nodes have not been found anywhere in the body, the retinal vessels are not involved, and the choroidal circulation is not visible.

Conclusions

A new disease, choriopathy, is placed on record. This is characterized by smoky, yellow-gray areas, which are widely dispersed, although at the onset only close to the disc, and increase in number and size from one-eighth to more than one-half disc diameter. They vary in shape, but are most often round or oval and show no visible migration of pigment. The fresh spots are most smoky and least defined. When once formed they persist, but become paler and more sharply demarcated. During the course of the disease a few, small, superficial retinal hemorrhages may develop in the macular region and also a smooth detachment of the lower portion of the retina, with clear subretinal fluid.

The cause is still undetermined.

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THE OCCURRENCE OF BLINDNESS IN NORWAY

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We must know exactly what we mean by the term blindness before we deal with its causes.

From a purely medical point of view, blindness means lack of perception of light. In ordinary parlance, persons lacking perception of light are said to be "totally blind." But there are many others who, from legal and practical social points of view, come under the term blindness. In most countries there are certain definitions with regard to the training and economic support of the blind. In paragraph 2 in the Norwegian law of July 16, 1936, concerning blindness insurance we find the following:

"According to this law, that person is blind who is incapable of vision or whose vision is so defective that he (she) cannot, after the correction of errors of refraction, find his way about by means of vision or cannot count fingers in good illumination against a dark background at a distance of more than one meter."

In Norway, there are no definitions with regard to the degree of vision of children who are referred to the school for the blind. This matter is settled on the merits of each case after a medical examination.

In Denmark, the child who cannot keep up with his school fellows because of defective vision is counted as blind and is referred to a school for the blind or a school for persons with defective vision. The Danes have no fixed definition of blindness in adults, but the Danish Blindness Association limits its membership to persons with vision of 4/60.

In Sweden and Finland, the definitions for children are the same as those in Denmark. In Sweden the limit for adults is 1/60; whereas, in Finland those persons rank as blind who cannot find their way about because of defective vision.

In Poland and Hungary also, the visual limit is 1/60. Other countries have similar definitions, but they are as a rule not so strict.

According to the English school law, those persons are counted as blind who are unable to read books used by children in national schools. The blindness law counts as blind the person who cannot see to do any work requiring eyesight. According to this law, a person is counted as blind when, after the correction of errors of refraction, his vision is less than 1/20 of the normal (about 3/60 with Snellen type). This criterion is, however, intended to serve rather as a general guide than as an absolute standard.

In Holland, a child is counted as blind when he cannot keep up with ordinary elementary school education because of defective vision. An adult is counted as blind when, because of defective vision, he cannot go out of doors without a companion or cannot see to count fingers three meters away. Thus the position is practically the same as that in England.

In France, the limit is put at less than 1/20. In Germany, it is put at less than 1/25 for children, and for adults, when the person concerned cannot find his way about, or his vision is 1/50 to 1/25, or he cannot count fingers two meters away. In Italy and Spain, industrial insurance includes the rule that vision of 1/10 or worse must be counted as blindness. The Spanish Blindness Association does, however, limit its membership to vision with finger-counting one meter away.

In the United States, vision under 20/200 is the official numerical standard for blindness. In Canada, it is 4/60.

It will thus be seen that definitions of blindness vary greatly, with limits ranging from finger-counting at one meter down to 1/10. The apparently rather arbitrary rul-

ings in this matter are determined, it would seem, by the obligations undertaken with regard to the blind by the various countries. But numerical standards do not provide a perfectly satisfactory limitation of our conceptions of blindness if we are to regard it from a practical social point of view which is incompatible with hard-and-fast limits determined by some definite degree of vision. For there are so many factors which are involved and which play a part in the employment of vision—the degree of any error of refraction there may be, the size of the field of vision, and so forth.

In the official definitions of several countries, account is taken of the extent of the field of vision. In practice, the patient's age plays an important part. A child without reading vision must be taught in a school for the blind even though walking vision may be good. But an adult accustomed to his work may often manage quite well even though his vision has gradually become so much reduced that he can no longer see to read.

The subjective conception of what is meant by blindness also varies. A person who has grown up as blind will always regard himself as blind even when his vision is fairly good, at any rate his walking vision; whereas, another person who in later life has gradually lost his vision will, on the other hand, often not speak of himself as blind, though only perception of light may be left to him.

Thus we see that the official definitions of blindness and the subjective conceptions of this term vary considerably, and it is therefore difficult to obtain reliable statistical data concerning the occurrence of blindness. It is also difficult to compare the reports on this subject in different countries. Any survey of the occurrence of blindness in a given country must include information concerning the acuity of vision of persons regarded as blind.

TABLE 1

AGE DISTRIBUTION OF THE BLIND

	Under 15 Years (percent)	15–65 Years (percent)	Over 65 Years (percent)
Hereditary	12.5	72.0	15.6
Diseases	1.0	81.4	17.5
Intoxications		96.0	4.0
Injuries	2.1	82.4	15.5
All	6.0	69.6	24.4
Age distribution in the population	22.6	68.8	8.6

The numerical data in the present review of the causes of blindness in Norway must be regarded as minimum figures. Complete information is lacking in some cases with regard to such items as the patient's age at the present time or when he became blind. In a few cases, the degree of vision is not mentioned. It is also quite certain that data concerning the patient's family are very incomplete, and this is the case with complications and so forth.

It appears from my investigations that in January, 1948, there were altogether 3,181 persons in Norway who must be considered as blind. Among these 3,181 were 1,838 men and 1,343 women (57.8 percent men and 42.2 percent women). This excess of men over women appears in most surveys of the frequency of blindness.

Table 1 shows the age distribution of the blind. Under the age of 15 years, there were 182 (about six percent) between the ages of 15 and 65 years there were 2,137 (69.6 percent) and over the age of 65 years there were 750 (24.4 percent).

The patient's age when he became blind is of just as much interest to us as his present age. Information on this score has been sought whenever possible, and it appears that, among the 2,925 cases in which the date of onset of blindness could be given more or less approximately, there were as many as 1,423 (48.6 percent) who were blind before the age of 15 years; whereas, there were 1,502 (51.4 percent) who became blind after this age. Among these were some (only 6.6 percent) who did not become blind till after the age of 65 years (table 2).

For the sake of comparison the tables in-

TABLE 2

AGE AT ONSET OF BLINDNESS

	Under 15 Years (percent)	15–65 Years (percent)	Over 65 Years (percent)
Onset of blindness	48.6	44.8	6.6
Age distribution in the population	22.6	68.8	8.6

clude the age distribution of the population as a whole. The figures referring to 1939, naturally, cannot be compared directly with the figures from 1948 or with the figures referring to the time at which a given patient became blind. But this comparison does, however, enable us to draw certain conclusions.

The figures showing the age distribution of the blind tell us in the first place that very many of them were thus afflicted early in life, nearly half of them before they were 15 years of age. We also see that blindness due to senile changes plays a smaller rather than a greater part among the other causes of it. Comparatively few persons become blind after the age of 65 years.

This statement is, however, made with a certain reservation—that in my material information concerning elderly folk who are blind in Norway is incomplete. For pensions for blindness are not given after the age of 70 years, and at this age the blind come under the ordinary old-age pension scheme with the result that there may be some persons who have become blind in old age and who do not come under any organization for the blind. Such persons may have escaped my notice. But I do not think that this state of affairs introduces any serious source of error.

At the present time, the age distribution of the blind relative to the age at which blindness sets in suggests that, as a rule, the blind live to be old.

We see that about half the blind had become so before they were 15 years of age. It is, therefore, obvious that the education of the blind and their subsequent fate present a social problem of far-reaching importance.

Table 3 deals with the degrees of blindness. As this table shows, 703 persons lacked perception of light, being "totally blind," whereas 789 were capable of perception of light but could not see to count fingers. Further, there were 443 with no better vision than finger-counting one meter away. Thus there were at least 1,935 (67.2 percent of the total) who were blind according to the definition of the Norwegian blindness law. Many of those with better vision than finger-counting one meter away suffered, however, from so severe a limitation of the field of vision that they could not get about, so it is certain that the number of persons blind in the legal sense was still greater than that given in the table.

Altogether there were 2,673 persons (93 percent of the total) with vision 5/30 or worse; that is, they could not see to read. Among the seven percent with vision better than 5/30, the overwhelming majority presented such great defects of the field of vision that they must be regarded as blind,

TABLE 3

VARIOUS DEGREES OF BLINDNESS

	Hereditary Diseases	Nonhereditary Diseases	Injuries and Intoxications	All	Percent
No perception of light	230	318	155	703	
No finger-counting	368	270	151	789	67.2
Fingers under 1 m.	204	189	50	443	
5/50	305	205	38	548	
5/30	124	53	13	190	25.7
Less than 5/10	114	36	6	156	
5/10	48			48	7.1
No reference	120	4		124	

TABLE 4

CAUSES OF BLINDNESS IN NORWAY (1948)

	Men	Women	Total
I. HEREDITARY DISEASES			
1. Anophthalmos	12	3	15
2. Microphthalmos	19	15	34
3. (a) Aniridia	15	10	25
(b) Coloboma iridis	13	14	27
4. Glaucoma infantile	30	23	53
5. Glaucoma juvenile	8	4	12
6. Luxated lens	21	12	33
7. Albinism	25	14	39
8. Atrophy optic nerve, congenit.	56	46	102
9. Atrophy optic nerve, Leber	10	2	12
10. (a) Heredodegeneration maculae luteae	6	4	10
(b) Laurence-Moon-Biedl's syndrome	3	2	5
(c) Familial amaurotic idiocy	7	4	11
11. Atrophy retina and choroid	44	33	77
12. Retinitis pigmentosa	155	108	263
13. Glioma retinae (and optic nerve)	4	7	11
14. Cataract, congenital	164	140	304
15. Amblyopia, congenital	14	11	25
16. Myopia, excessive	37	57	94
17. (a) Amotio retinae+myopia (one eye)	10	13	23
(b) Amotio retinae+myopia (both eyes)	43	35	78
(c) Amotio retinae without myopia	30	7	37
18. Degeneration of cornea	1	3	4
19. Keratoconus	3	3	6
20. Various diagnoses	10	7	17
21. Glaucoma simplex	89	56	145
22. Glaucoma, inflammatory	3	2	5
23. Cataract, senile	12	21	33
24. Uncertain diagnosis	49	36	85
TOTALS	893	692	1,585

	Men	Women	Total
II. NONHEREDITARY DISEASES			
A. DISEASES			
1. Blennorrhea neonatorum	32	28	60
2. Keratitis parenchymatosa	24	46	70
3. Sclerokeratitis	98	162	260
4. Iridocyclitis	50	60	110
5. Uveitis	44	55	99
6. Retinitis:			
(a) Chorioretinitis disseminata	19	16	
(b) Chorioretinitis and congenital lues		4	
(c) Chorioretinitis centralis	6	2	
(d) Degeneration maculae lutea, senile	3	9	
(e) Periphlebitis retinae	7	3	
(f) Retinitis proliferans (+ exudativa)	13	2	
(g) Retinopathy hypertonica, nephritica	5	13	
(h) Retinopathy diabetica	17	6	
			125
7. Atrophy optic nerve			
(a) Tumor cerebri	28	29	
(b) Encephalomeningitis	26	23	
(c) Neuritis optic nerve	12	16	
(d) Lues, congenital	11	7	
(e) Lues, acquired	44	7	
(f) Apoplexia cerebri	5	3	
(g) Various causes	6	1	
(h) Unknown causes	34	13	
			265
8. Two of diseases listed above	6	2	8
9. Various diagnoses	12	13	25
10. Uncertain diagnoses	53	65	118
TOTALS	555	585	1,140
	Men	Women	Total

TABLE 4—*continued*

B. Intoxications			
1. Atrophy optic nerve, alcoholic + nicotin	7		7
2. Atrophy optic nerve, methanol	42		42
3. Atrophy optic nerve, gas poisoning	1		1
	50		50
C. Injuries			
1. Atrophy optic nerve, traumatic	12	2	14
2. Direct injuries	193	14	207
3. Sympathetic ophthalmia	105	45	150
4. Injury one eye, disease the other eye	30	5	35
	340	66	406
Summary			
I. Hereditary diseases	893	692	1,585
II. Nonhereditary diseases:			
A. Diseases	555	585	1,140
B. Intoxications	50		50
C. Injuries	340	66	406
Totals	1,838	1,343	3,181

at any rate from a practical social point of view.

Causes of blindness

I have classified the causes of blindness in two main groups: (1) The diseases supposed to be hereditary and (2) those which are not presumably hereditary. The figures dealt with in my survey are distributed in practically equal numbers between these two groups, with 1,585 cases of hereditary blindness and 1,596 cases which were not hereditary.

In the overwhelming majority of cases the diagnosis depended on examinations by oculists.

That ailment which is taken to be the primary factor is to be put on record as the cause of blindness. In many cases congenital defects of the eyes lead sooner or later to serious complications which may entail total blindness. For example, glaucoma, amotio retinae, or cataract may develop as a complication of aniridia, an ectopia lentis, retinitis pigmentosa, and so forth. In such cases the original congenital defect is given as the cause of blindness.

In Table 4 the various diagnoses are more fully specified. We see that males are in the majority (57.8 percent males as compared with 42.2 percent females). This excess of males depends primarily on the relatively high rate of trauma among them (there were 340 males and only 66 females whose blindness was due to trauma).

Certain hereditary diseases are also more common among men than women—luxated lens, albinism, retinitis pigmentosa, atrophy of the retina and choroid, Leber's atrophy of the optic nerve, congenital cataract, amotio retinae, and simple glaucoma. The only hereditary disease to which women are definitely more prone than men is excessive myopia.

It will be seen that the sex distribution of the nonhereditary diseases varies greatly, women being in the majority in the case of sclerokeratitis, parenchymatous keratitis, iridocyclitis and uveitis; whereas, men are much in the majority with regard to retinitis and, in particular, atrophy of the optic nerve. But on the whole there are a few more females than males in this group.

Brief mention may be made of some of the most important diagnoses, and a few odd points of special interest will be emphasized.

Hereditary causes

Glaucoma infantile

Of the 53 patients, 30 were males and 23 were females. In 45 cases, there was no other such case known in the family—a point indicative of a recessive disease. Among these 53 patients there were only six over the age of 40 years. This is quite remarkable. It is most improbable that such patients should show any increased death rate, and it is more likely that the scarcity of cases over the age of 40 years reflects uncertainty over the diagnosis.

For in most cases, the blind eye suffering from infantile glaucoma becomes more or less changed with age with the result that a retrospective diagnosis may be difficult. So it may happen that some elderly patients now coming under the heading of sclerokeratitis originally suffered from congenital glaucoma, their eyes having shrunk so much or undergone some other change, having perhaps been enucleated, that an accurate diagnosis is no longer possible.

Glaucoma simplex

There were 89 men and 56 women in this group, the sex ratio being the same as that observed elsewhere in Scandinavia. Further south, the male majority is not so evident, and women seem often to be in the majority. Nearly all these cases were isolated, and there were only 13 in which there was a history of glaucoma elsewhere in the family.

On the whole, the data concerning the families of these patients were scanty, and they do not justify us in drawing any conclusion with regard to the influence of heredity; but it is assumed that the process is dominant. Every ophthalmologist in practice is familiar with the fact that glaucoma simplex often occurs as a familial disease, to some extent in direct sequence, from one generation to another.

Atrophy of the optic nerve

This diagnosis covers many different forms of disease difficult to separate into definite groups, partly because there are presumably many transitional forms, and partly because it is difficult to obtain complete information about the history of the course of the disease and precise information derived from examinations of the eyes. The cases dealt with here were presumably of the hereditary form.

Altogether there were 114 such hereditary cases of blindness, and among them were 12 typical cases of Leber's atrophy of the optic nerve. They occurred in two families, with five cases in one family and seven in the other. It is assumed that Leber's optic-nerve atrophy may be a sex-linked recessive, whereas the ordinary hereditary optic nerve atrophy seems to be a simple recessive.

Atrophy of the retina and choroid

This diagnosis is rather vague and probably covers several different forms of disease. Further, there are certain atypical forms which may possibly belong to other groups such as retinitis pigmentosa for example. There were altogether 77 (44 males and 33 females) persons whose blindness qualified them for inclusion in this group.

The disease was familial in fully half of these cases. It seems to be transmitted as a simple recessive disease.

Retinitis pigmentosa

This is one of the most common causes of blindness in Norway. Of the total of 263 persons, 155 were males and 108 were females. This preponderance of males is the usual condition, and the ratio of 3 to 2, as in the present instance, is to be found in many other statistics.

It is easy to recognize typical retinitis pigmentosa, but there are many atypical forms in which the diagnosis may almost be said to be a matter of personal opinion. The overwhelming majority of the cases in the present instance were typical retinitis pigmentosa cases.

Both the typical and atypical cases may in

TABLE 5

RETINITIS PIGMENTOSA

	Familial Cases		Isolated Cases	All
	Number of Affected Individuals	Number of Marriages		
Parents related	58	28	17	75
Parents not related	65	29	27	92
No information concerning relationship	36	16	60	96
Totals	159	73	104	263

some instances occur in one and the same family and, when this was so, I have counted them all under the heading: Retinitis pigmentosa. A few atypical cases, not suitable for inclusion in any of the other groups, have been placed in this group. It is particularly in connection with atrophy of the retina that the transition between it and retinitis pigmentosa may be very ill defined.

In 104 cases of retinitis pigmentosa the disease was not known to have occurred in any other member of the same family. In some instances it was stated that "blindness" occurred in the family. In these instances, and when there was no further information on the subject, it was assumed that the cases were isolated, that is, only one in each family.

There were 73 families in each of which there were several cases of retinitis pigmentosa, usually observed in several siblings, but also in more distantly related members of the family. There were 15 families with three cases in each, and these patients were usually siblings. There were 17 families with four cases each, two families with five cases each, and one family with eight cases of retinitis pigmentosa.

The following observations were made with regard to blood ties between the parents: Among the familial cases there were 58 in which the parents were consanguineous, and among the isolated cases there were 17 in which this was the case. Thus there were altogether 75 patients with consanguineous parents (table 5).

It will be seen that information is most complete with regard to the familial cases, and it is quite natural that the persons concerned took a greater interest in the matter and were more willing to supply information about their relatives. It is probable that marriages between blood relatives were even more common than this table shows. But the fact that more than 25 percent of these patients had consanguineous parents is strong evidence indicative of a recessive process in the transmission of the disease.

There was one family in which the process was definitely dominant. In this case, a man suffering from retinitis pigmentosa was the father of eight children, six of whom developed retinitis pigmentosa. One of the afflicted daughters gave birth to five children, the eldest of whom, at any rate, also contracted this disease.

In some cases, the hereditary mode of transmission of the disease is more in doubt. In one case it was stated that the mother and a sister had very defective vision, but I have unfortunately been unable to obtain further information about them.

In another family it was stated that the father and grandmother of two brothers suffered from impaired vision. It was also stated about a woman suffering from retinitis pigmentosa that her father suffered from a similar disease; whereas, the vision of her two children (not yet grown up) was good. In another case, four siblings and a grandfather were said to have the same disease, without the existence of any blood tie between the parents.

I have been unable to find any example of sex-linked hereditary transmission of reti-

nitis pigmentosa, but my material is far from being completely studied from the genetic point of view. Deaf mutism and retinitis pigmentosa often occur together, and 22 of the blind persons in my study were also deaf. This is almost certainly an underestimate. Idiocy was reported in 11 cases, epilepsy in five, insanity in four.

The age distribution of these cases of retinitis pigmentosa was as follows:

Age	Years	Cases
Under	15	6
Between	15 and 19	17
Between	20 and 69	229
Over	70	20

This is what one would expect with a disease which usually makes its first appearance at a juvenile age. First there are only a few children, and then we find an accumulation of cases at a slightly older age. It is often difficult to find out the age at which blindness began in cases of retinitis pigmentosa, data on the subject being frequently rather vague. "Impaired vision increasing since the school age" is a very common statement or "steadily decreasing vision." But in some cases information on the subject was more precise, such as: "Unable to read during the last 10 years," and so forth.

According to available information, the following figures show when blindness overtook these patients suffering from retinitis pigmentosa:

Age	Years	Cases
At birth or before	5	62
Between	5 and 9	32
Between	10 and 14	20
Between	15 and 19	28
Between	20 and 39	59
Between	40 and 49	17
Between	50 and 59	5
Over	60	1

In 22 cases, vision had "gradually diminished," but nothing was said about the age at which this happened.

The actual degree of loss of vision in these cases of retinitis pigmentosa does not give us a correct impression of the degree of invalidism suffered. For it is the limitation of the field of vision and the hemeralopia which render these patients so helpless. But the central vision in most of these cases, totalling 123, was, however, much reduced, with inability to count fingers. There were 60 other patients who could not count fingers more than one meter away. In nearly all the cases in which vision was better, the field of vision was much reduced with the result that these patients could no longer see to walk.

Congenital cataracts

Of the 304 blind persons in this group, 164 were males and 140 were females. In 184 cases—that is, in three-fifths—there was only one such case in each family; whereas, in the remaining 120 cases, each family presented two or more such cases. For want of information on the subject, it is possible that some of the cases which I have counted as isolated were in reality familial cases. For it often happened that information about a given family was very incomplete or was vague, with indefinite statements about "blindness in the family," parents with defective eyesight, or siblings or other relations in the same category. For want of more accurate information, I have not in such instances assumed forthwith that the cause of the defective vision referred to was identical with that found in a given case.

In 22 families there was a total of 50 cases of congenital cataract distributed over several generations. As a rule, precise information was obtained only with regard to two generations. In two families with reliable information as to the occurrence of this disease in three generations, it would seem that the hereditary transmission of the disease was plainly dominant. In other families it seems rather to be recessive, witness the fact, among others, that in many cases the parents were blood relations.

The information available was in most cases too scanty, however, to warrant the drawing of definite conclusions concerning the hereditary mode of transmission of the

disease. Altogether, in 18 cases the parents were blood relatives.

Information was forthcoming with regard to the treatment which 229 of these cases of congenital cataract had received. In some cases various reasons were given as to why there had been no treatment (for example, complications which had contraindicated treatment). In a few cases the patient had refused treatment, and in others there was, perhaps, enough vision without the institution of any treatment other than the administration of mydriatics.

Among the patients operated on, 209 had operations on both eyes, and 20 on only one eye. As a result of bilateral operations, vision was worse than 5/50 in the best eye in 130 cases, and better in 79 cases; in monocular cases, it was worse than 5/50 in 16 cases and better in four cases.

The reason why only one eye was operated on was, as a rule, that the other eye presented complications contraindicating an operation or that the operation on the first eye had given such poor results that the patient preferred not to risk an operation on the other eye.

Among the 253 cases of congenital cataract about which information concerning vision was available, there were 193 with vision worse than 5/30. In other words, they could not see to read, and at least 101 of them could not see to walk.

Even though congenital cataract may in a few cases be a comparatively benign ailment entailing little invalidism, it means in the vast majority of cases a very great reduction of vision.

Excessive myopia

This is perhaps a group which is not quite uniform, but in the present instance it includes only "pure" cases as far as possible—that is, cases in which the myopia was very marked and had been much in evidence since earliest childhood.

The lowest degree of myopia recorded among the refracted cases was 10D. In other cases, the degree of myopia was vaguely expressed as "of high degree," "severe," "myopia magna," and so forth. In most cases there were degenerative changes in the fundus in addition to the myopia, but they were not regarded as part of any primary lesion of the anterior part of the uvea, being interpreted rather as a complication accompanying the myopia.

There were altogether 94 blind persons with the diagnosis of excessive myopia (37 males and 57 females). This preponderance of females tallies with that found by others (Blegvad) in cases of excessive myopia. But myopia is in my experience the only hereditary cause of blindness with a preponderance of females.

Nothing was known with certainty with regard to other myopic members of the same family in 70 cases. In 24 cases there was more than one known case in the same family, and in nine cases at any rate, the parents were known to be blood relatives.

The great number of isolated cases and the frequency of consanguineous parents are suggestive of a recessive process, even though in a few cases there may also have been a possibility of a dominant process.

Amotio retinae

This condition is closely related to excessive myopia as it is so common among myopic patients, particularly when the myopia is excessive, though even the lower grades of myopia may to a great extent be associated with detachment of the retina.

There were 140 blind persons in this group (84 males and 56 females). The disease was isolated in 112 of these cases, whereas in the remaining 28 it was distributed among 14 families. Detachment of the retina happened to the members of several generations in some cases, and in at least four cases one of the parents and one of their children presented this disease. In 10 more cases it was stated that one of the parents was nearsighted.

In the present material it is difficult to

form any opinion as to the hereditary mode of transmission of the disease. In five cases trauma was said to be the cause of detachment of the retina on one side, whereas it was "spontaneous" on the other side.

In the overwhelming majority of cases detachment of the retina occurred somewhat late in life, at the age of 40 or 50 years, as an expression of early senile changes. To some extent the hereditary transmission of this disease depends on anatomic conditions which have been inherited, particularly slight abnormalities of the blood vessels in the fundus where the nutrition of the retina may be interfered with at certain points, and there may be an increased predisposition to the formation of a retinal hole.

Causes of blindness: Nonhereditary

The classification of the diseases of the eyes which are not hereditary has depended primarily on principles concerned with pathology and anatomy and not with etiology. For the etiology of many of the diseases of the eyes is far too uncertain. Besides, the information available with regard to many cases in the present material was so incomplete concerning such matters as serologic reactions, tuberculin reactions, and the like that no definite conclusions could be drawn. However, the first two groups, dealing respectively with blennorrhea neonatorum and parenchymatous keratitis represent, in reality, diagnoses in which the etiology, as well as the pathology and anatomy, is at once apparent.

Blennorrhea neonatorum

Of the 60 blind persons in this group, 32 were males and 28 were females. The diagnosis was, perhaps, not quite certain in every case. The available information was to some extent a little vague with such terms as: "Inflammation in both eyes just after birth," "large central leukomas—born out of wedlock," "blind in infancy," "milk-white corneas," and so forth. In most cases the eyes had shrunk subsequently, and some of them had been removed for the relief of pain. It is conceivable that a case or two of congenital glaucoma may be hidden in this group. An overlooked trauma or phlyctenular ophthalmia may also possibly have been responsible for a few of these cases.

Not one of the persons whose blindness was due to blennorrhea neonatorum was under the age of 20 years at the time of the present investigation. Six were between the ages of 20 and 30 years, 10 were between the ages of 30 and 40 years, and 44 were more than 40 years of age. Thus, as was to be expected, there was a marked upward shift in the age distribution of these cases.

As these figures have nothing to do with the incidence of blennorrhea neonatorum, and only with the number of persons rendered blind by it, we cannot be sure that it was Credé's prophylactic procedure with silver nitrate or the more effective treatment of this condition during the last decades which is responsible for the encouraging reduction in the incidence of blindness as the result of a disease which can so well be avoided. It is probable that both these factors have played a certain part.

Keratitis parenchymatosa

Of the 70 blind persons in this group, 23 were males and 47 were females—a preponderance of females which tallies with the findings of large collective investigations. In most of my cases the diagnosis was based on a Wassermann test, but some of the patients were so old that they belong to the period without serologic tests. It is therefore possible that in one or other of these cases the blindness was not due to congenital syphilis. There was one case in which the blindness was presumably due to acquired syphilis, the patient having contracted this disease at the age of 18 years, and having developed parenchymatous keratitis at the age of 34 years.

With regard to the age distribution of these patients, there was none under the age of 15 years, and only one between the ages of 15 and 19 years. There were 17 between

the ages of 20 and 29 years, and 52 over the age of 30 years. On the other hand, the age distribution at the time of the outbreak of the disease showed the usual arrangement, with the bulk of the cases in the first and second decades of life, the onset of the disease being before the age of 20 years in as many as 55 cases, or fully 71 percent of the total.

Miscellaneous

Each of the following groups: *sclerokeratitis, iridocyclitis* and *uveitis,* as well as *retinitis* and *atrophy of the optic nerve* is so heterogeneous that it is impossible to go into any great detail in the present study. These groups play a very big part in the causation of blindness, however, for there were altogether 880 persons whose blindness was due to one or other of these diseases.

There were no women among the persons whose blindness was due to *intoxicants or poisons.* The most important agents from a practical point of view were methanol (42 cases) and tobacco and alcohol (seven cases).

In the 1930s there were three persons whose blindness was due to methanol. Between 1941 and 1945 there were 37 rendered blind by it, and after 1945 there were only two such cases. This improvement may presumably have been due in part to educational propaganda with regard to the dangers of methanol consumption, and in part to more effective treatment instituted of late years as soon as methanol poisoning was suspected.

Trauma

There were 93 cases of blindness due to mining accidents among adults and 29 cases of percussion-cap accidents among children. In 26 cases the blindness was directly due to the war, and 13 of them were the outcome of the explosion disaster in Bergen on April 20, 1944. Seven insane persons had blinded themselves either by repeated blows of the fist on the face or by wrenching their eyes directly out of their heads.

Sympathetic ophthalmia

Of the 150 persons in this group, 105 were males and 45 were females. The injury responsible for inflammation in the first eye concerned children in two thirds of the cases and adults in one third. In nearly all the cases in which information on the subject was available, the sympathetic ophthalmia developed within a year of the injury, in most cases within the first half year.

Discussion

On closer scrutiny of my material I have made many observations of such interest that they deserve closer attention. Here are a few points of special interest:

In the first place, it is remarkable how many of the blind were mentally defective and backward—a total of 160 persons. Their distribution between the two main groups, the hereditary and the nonhereditary, was also curious. As many as 133 belonged to the hereditary group, while there were only 27 whose blindness was due to some acquired lesion. This observation is, by the way, neither new nor very remarkable, for it merely stresses the fact that the eye is an advanced part of the cerebrum, and a faulty development of the latter therefore often entails involvement of the eyes.

The blind who were insane numbered 11 in the hereditary group and 22 in the nonhereditary group. This preponderance of cases in the latter group is presumably due in part to the fact that altogether seven of these persons became blind as a direct consequence of their insanity which led to self-mutilation, in part to the difficulty encountered in treating diseases of the eyes in some cases of insanity. In some such cases it may even be impossible to give the necessary treatment such as an operation for cataract, for example. Further, in some cases a disease of the brain may give rise both to insanity and to atrophy of the optic nerve.

We find the familial occurrence of blindness much accentuated in the case of the hereditary diseases, for 45 percent of my

hereditary cases were familial, whereas the corresponding figure for the nonhereditary diseases was only 6.5 percent. Indeed, the familial occurrence of blindness in the nonhereditary group depended, among other things, on such facts that congenital syphilis may attack several children in one and the same family, and tuberculous diseases may also prove to be familial.

No reliable information exists either in Norway or elsewhere with regard to the frequency of consanguineous marriages in the population as a whole, but Gunnar Dahlberg suggests that in the countries of Western Europe about 0.5 percent of all marriages are between cousins.

My own investigations of blindness in Norway show that consanguineous marriages are remarkably common among the parents of blind children. But I have also found that the frequency of such marriages differs greatly according as blindness is due to hereditary or nonhereditary diseases.

In the hereditary group, there were consanguineous marriages in 173 cases, whereas in the nonhereditary group this was the case in only 26 marriages. It is a little difficult to make a calculation on a percentage basis because I do not know what was the total number of marriages. If we assume that there was only one blind person for each marriage, we shall find that the marriages were consanguineous in about 11 percent of the cases of blindness in the hereditary group, while the corresponding figure for the nonhereditary group was only about 1.6 percent.

As it is particularly in the group of hereditary diseases that we often find several blind children from one and the same marriage, the number of consanguineous marriages to be counted becomes comparatively small. In other words, the percentage of diseases due to heredity is in reality considerably higher.

Lastly, what is to be done to treat or prevent blindness? It is probable that certain of the nonhereditary diseases will lose some of their importance with the progress to be expected in the development of the new chemotherapeutic drugs and antibiotics. Here there seem to be many possibilities in view, though we cannot expect any radical readjustment of our therapeutic principles.

With regard to the hereditary diseases we must in the future seek to intervene, in a far greater degree than hitherto, with prophylactic measures, that is, the prevention of a defective progeny. By such measures it should be possible to prevent the tragedy represented by the advent of one blind child after another in the same family.

A well-known blind man in Norway has written on this subject: "In my opinion all should be done than can be done by the community with a view to preventing the birth of blind children into the world. To go blind through life is an all too great price for a child to pay for the infatuation of its parents."

It is evident from what has just been said that the hereditary forms of blindness occur so frequently and at so young an age that they indeed represent a social problem. It would therefore be of great importance if something could be done to reduce the number of cases of hereditary blindness in the future.

To date it has chiefly been the treatment of existing diseases which has attracted attention. Unfortunately, however, these diseases do not react at all or only to a slight extent to the treatment hitherto available. To be sure, during the last 50 years great advances have been made—for example, in the treatment of congenital cataract, the various forms of glaucoma, and detachment of the retina. Yet in spite of the treatment they receive, very many patients must be counted as blind because of some hereditary disease.

There is a little chance, alas, of the *treatment* of these and other hereditary diseases of the eyes giving appreciably better results in the future than they do at present. As a rule, these diseases are complicated by other ailments of the eyes, and it is often these other ailments which are responsible for

defective vision and not the immediate effects of an operation which may have been technically successful.

Here we are confronted by a very important question: Can anything be done to *prevent* the hereditary diseases of the eyes? Can eugenic advice be helpful here? Although something has been done in this field, it has been done only in isolated cases. Here I would discount the exaggerated measures, to some extent totally lacking any scientific basis, adopted in Nazi Germany where the attitude to this problem was most unfortunately discredited to such an extent that it may be difficult to revive interest in it again.

In this connection, I would like to quote from Duke-Elder's textbook, volume 3, page 2767):

> When it (hereditary disease of the eyes) appears as a *dominant* characteristic each affected individual gets the disease directly from one parent and the chances are that it will be passed on to half the children. Unless it is considered right to produce children who will eventually become blind, these people should have none.

If we agree with these sentiments, as most of us surely do, the question arises: How are we to frame a correct policy? There is no point in adopting only general principles such, for example, as the prohibition of all consanguineous marriages or the categorical refusal to permit marriages between persons with a family history of hereditary blindness. Here, as everywhere else in medicine, each case must be taken by itself.

What is now of importance is to arrive at as accurate a diagnosis as possible, not only with regard to the individual case, but also the hereditary character of the disease as far as possible. We all know that there are many diseases whose appearance and behavior seem to be quite uniform, although the features determining their hereditary transmission may be different, as in the case of retinitis pigmentosa and congenital cataract, both of which may present dominant or recessive hereditary features.

This is why the study of a case by itself is insufficient; we must have an accurate family history enabling us to decide which particular feature characterizes the hereditary transmission of the disease. As a rule, it is not the duty of a doctor by himself to collect information about the occurrence of diseases of the eyes in a given family, and it is therefore desirable for all the necessary data to be collected in a central office in a card index.

Once definite information is available concerning several generations in a given family, it will be easier to decide how hereditary diseases are transmitted. The further back such a card index goes, the greater will be its value. It will enable the doctor who is consulted on the subject to obtain information which the patient himself can hardly be expected to possess so accurately that it is of sufficient value.

As a rule, all the patient knows is that some relative or other has defective eyesight, or is blind or suffers from some ailment like his own. The more accurate the information at the disposal of the doctor, the better will he be able to advise his patients with regard to such procedures as sterilization.

A complete medical registration of the blind is therefore an important weapon in the campaign against hereditary blindness. Here it should be emphasized that such registration should not only include persons who are blind in the legal sense, but also those who suffer from the various ailments which in different degrees may overtake the eyes such as congenital cataract, retinitis pigmentosa, various forms of glaucoma, albinism, and so forth. Even within one and the same family the behavior of these diseases may vary. Registration of every case is of importance in deciding what is the particular mode of transmission of the disease. To insure accurate family records it is, of course, necessary to possess information about the healthy members of the family, at any rate their number and sex.

There is, to be sure, little we can prophesy with certainty concerning the influence of heredity in human beings, but there are some cases in which it should be possible to give

well-grounded and effective advice when the character and hereditary features of a given disease are well known.

Most of us probably agree with Duke-Elder in his opinion that persons subject to dominant inheritance of blindness should not beget children, and that consanguineous marriages should be avoided if there is a recessive form of blindness in the family. When there is no blood tie, but there is the same recessive disease in the families of two persons contemplating marriage, the problem is the same and they would do well not to beget children.

It is more difficult to make a decision when it is known that blindness in a recessive form occurs only on one side of the two families; whereas little is known about the hereditary conditions in the other family. Here it is impossible to lay down the law with certainty. So much depends on the character of the disease and on its gravity which may, perhaps, vary considerably in different families or in different generations. There may also be other factors to consider in deciding for or against procreation in a given case.

Sometimes a crop of cases of blindness makes its appearance among the siblings in certain families in which nothing is known of hereditary disease of the eyes. In such cases we must assume that both parents are heterozygous carriers of a recessive disease, and if it is typical it should be possible to hinder a further outbreak among the siblings. We are, however, confronted here by the difficulty that the disease often manifests itself for the first time rather late in childhood with the result that several children may be born into the family before the disease is recognized.

It is therefore necessary when a hereditary disease has been discovered in a child to examine carefully its brothers and sisters, still apparently well, in order to throw as much light on the problem as possible. If we now find that several children present the same disease, that it has great "penetrative effect," the advent of more children should at any rate be prevented.

I do not propose to specify the procedure to be adopted for each hereditary disease. We should not entertain any exaggerated hopes with regard to the effects of applied eugenics on the hereditary causes of blindness from a purely statistical point of view, but *something* can surely be done. To succeed, it is necessary, to begin with, that both the doctor and the patient should be aware of the hereditary character of the disease.

The blind often try to explain away the hereditary character of their blindness, and they find all sorts of reasons for showing how the disease broke out. If the doctor is to be sure that the disease is hereditary, his examination must be thorough, and he needs information about the family if he is to find out how the disease is transmitted.

Educational propaganda concerning the hereditary diseases of the eyes should be as effective as possible, and the public authorities should provide for the registration of all who suffer from those diseases of the eyes which may lead to blindness and which may be regarded as hereditary.

Such registration would, of course, also be of value in the campaign against the non-hereditary diseases which may lead to blindness; it will enable us to follow the rise or fall of the incidence of the various diseases of the eyes and thus to gauge the effects of the campaign against blindness. It will also possibly give us clues to the improvement of this campaign in the future.

Pilestredet 28.

SIMPLE PRACTICE METHODS FOR INTRACAPSULAR CATARACT EXTRACTION

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Dr. Arnold Knapp in his introduction to *Surgery of the Eye* by Török and Grout said: "Operations on the eye demand first of all much practical exercise to acquire the proper technique in the manipulation of the instruments. This can only be accomplished by practice, observation, and suitable instruction."

Practice is essential, not only for the tyro but also for the experienced surgeon who continually strives for perfection in each maneuver. As Michelangelo said: "Trifles make perfection and perfection is no trifle." A little practice each day is of more value than long periods of practice at irregular intervals. The surgeon, as the pianist, must practice regularly in order to maintain his high degree of manual dexterity. Even the great Paderewski is said to have practiced many hours daily during concert tours to achieve perfect performance.

The eyes of the fresh cadaver are undoubtedly the best material upon which to practice but they are not available to most ophthalmologists in sufficient numbers to be practical for this purpose. Eyes of animals, the use of which are familiar to all ophthalmologists, are next in order as suitable practice material. When animal eyes are available, practice upon them is superior for most procedures to the methods described in this paper. To practice the section, a cadaver or animal eye is essential.

There are, however, many other steps in the cataract operation that can be practiced without the use of eyes. Textbooks give but little instruction in methods of acquiring skill in the use of instruments and development of surgical technique. It is the intention of this paper to describe a few simple practice methods.

The surgeon should, first of all, be familiar with his instruments and able to hold them lightly with fingers and hands relaxed and in a comfortable position. The use of instruments can be easily practiced without cadaver or animal eyes by simulating the motions employed in operations upon the human eye.

To become skillful with either hand in the use of scissors and other instruments requires considerable practice. Cotton, paper, or any soft material may be used to practice the use of scissors. Eye scissors can be carried in a pocket and, in odd moments, one can practice holding and turning them, as is so often necessary when using curved scissors.

To hold fixation forceps, particularly those with a catch, in such a way that the catch can be released easily without traction or pressure, can be mastered without the use of eyes. The same is true of the needle driver, one can practice holding it so that the thumb or finger is always in the proper position to release the catch easily without a snap or movement of the instrument. Deft handling of this instrument adds greatly to the safety and smoothness of suturing.

Even when the accomplished surgeon changes from regular- to cross-action forceps, particularly cross-action capsule forceps, some preliminary practice is essential.

To practice suturing, all that is required is the needle driver, a pair of forceps, needle, thread, and material to suture. Tissue paper, such as kleenex, is excellent for some types of suturing because it tears easily with slight traction and traction should be avoided in most instances. To be able to hold the lip of the wound without traction or pressure is of sufficient importance to warrant practice.

For suturing more nearly comparable to the corneoscleral suture, a small rubber ball, solid, or hollow, may be used. An "Asepto" syringe bulb about one inch in diameter is excellent for this purpose. A heavy suture in-

troduced at the pole opposite the opening serves in lieu of the optic nerve to be held by the clamp of the manikin (fig. 1). A second suture may be placed at the usual site where the globe is grasped for fixation, as it is difficult to grasp the smooth bulb or ball unless sharp toothed forceps, similar to the Elschnig forceps, are used. An incision is made in the bulb, partial or complete, depending upon the type of suture to be practiced.

Fig. 1 (Atkinson). "Asepto" syringe bulb, showing sutures—one at posterior pole for clamp of manikin and one for fixation.

Fig. 2 (Atkinson). Bulb in manikin.

If a postplaced suture is to be practiced, interest is added if the bulb is partially filled with water, nearly to the incision. One can then try to introduce the sutures without spilling the water.

An advantage in using the rubber ball instead of an eye is that, once it is arranged in the manikin, it is always available for practice. A block of wood with a hole in it is more convenient than the usual manikin as it can be easily slipped into a desk drawer

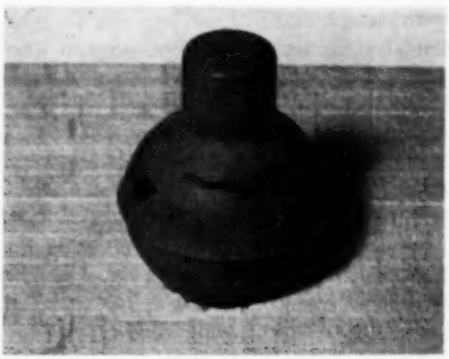

Fig. 3 (Atkinson). Bulb in block of wood.

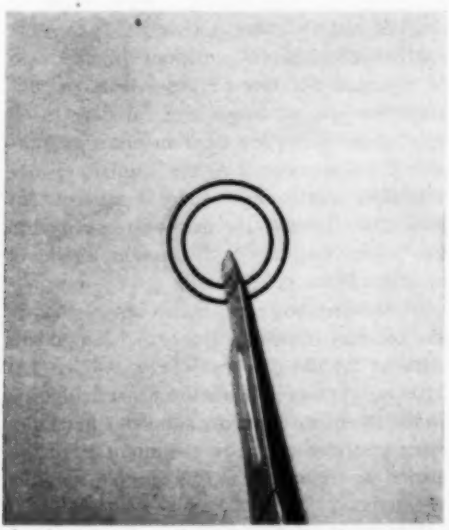

Fig. 4 (Atkinson). A superficial cut is made and a ridge turned up consisting of the superficial layer of the paper.

(figs. 2 and 3). The manikin with instruments may be left in some convenient spot so that a few minutes of practice can be had at odd times without the inconvenience of preparing eyes and cleaning up afterward.

One of the most important procedures to master in the intracapsular extraction is the proper coördination of traction and pressure used to dislocate and extract the cataractous lens. A three-by-five-inch scratch pad furnishes an excellent means by which to practice the use of the capsule forceps and hook or whatever instrument is used to exert pressure.

Two circles are drawn on the lower part of the sheet, one inside the other with the first circle the size of the cornea and the inside circle representing the dilated pupil. At the site where the capsule is to be grasped by the forceps, a superficial cut is made in the paper and a ridge consisting of the superficial layer of the paper is turned up (figs. 4 and 5).

Capsule forceps are then used to grasp the capsule in a manner similar to that used to grasp the capsule in a human eye. Closed forceps are slid gently along the surface of the paper as if introducing them into the anterior chamber. When the ridge is reached, the forceps are opened, gentle pressure is exerted on the pad, and the small ridge of paper is grasped with the forceps. Slow, deliberate movements from side to side are made with the forceps and the paper is lifted a little, as is done to dislocate the lens. As the paper is raised slightly, pressure is exerted with the hook (fig. 6).

With some imagination one can go through the maneuver of dislocating and extracting the lens. Slight tearing of the fragile ridge of paper or the pulling loose of even a single wood fiber of the paper can usually be felt. In this way, since the slightest tear can be felt, a delicate tactile sense is acquired and, with practice, the sheet of paper can be lifted and moved without tearing the small tag or ridge.

For practice with the erisophake, a piece

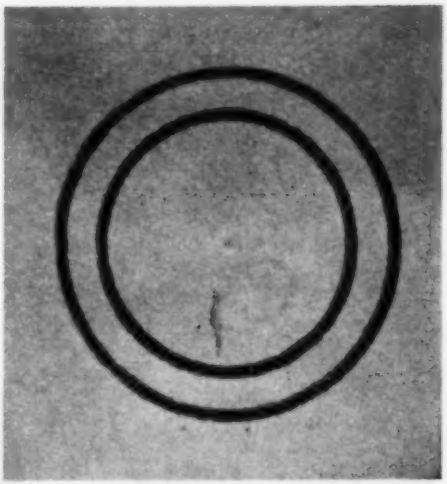

Fig. 5 (Atkinson). Shows the ridge.

Fig. 6 (Atkinson). The forceps grasping the ridge and pressure exerted below with the hook.

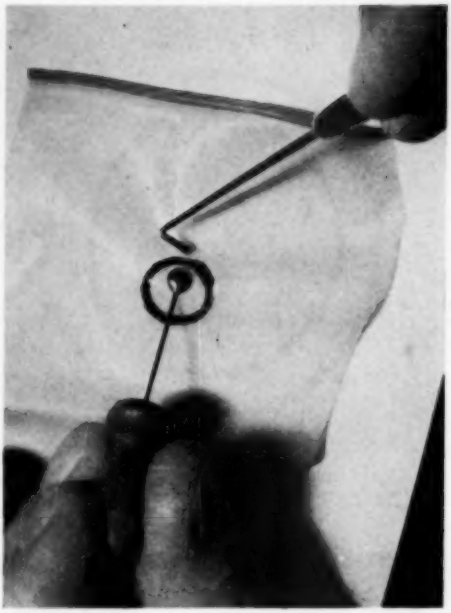

Fig. 7 (Atkinson). A piece of rubber from an old rubber glove for practice with the erisophake.

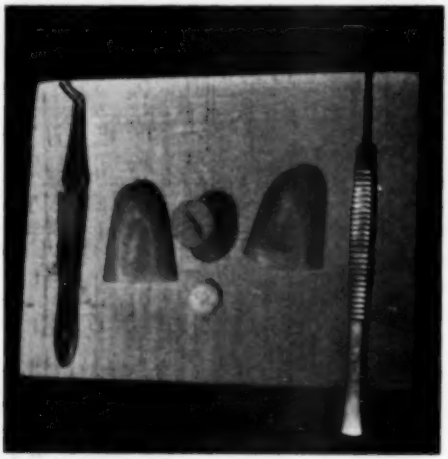

Fig. 8 (Atkinson). Cork in block of wood, two finger tips from discarded rubber glove, the yarn-covered button which serves as the cataract, forceps, and hook.

of rubber from an old rubber glove may be used in the same manner as the paper (fig. 7). The erisophake grasps the rubber

Fig. 9 (Atkinson). Extraction of cataract, head first.

Fig. 10 (Atkinson). Extraction of cataract by tumbling.

better if the surface is wet. Good coördination between the two hands can thus be developed so that fewer capsules will be ruptured and less vitreous lost.

Another way to practice the extraction is with a small button about the size of a cataractous lens covered loosely with yarn to serve as the cataract. For the practice eye, a cork about 15 mm. in diameter may be used. The top is hollowed slightly to make it

concave. Over this is stretched a finger-tip of a discarded rubber glove. This layer of rubber over the top of the cork represents the hyaloid on which is placed the yarn-covered button.

A second finger-tip which is to serve as the cornea is drawn loosely over the first so that a space is left large enough for the yarn-covered button. The convexity of the finger-tip simulates the curve of the cornea. The cork can be inserted into a hole in a small board or clamped in a manikin. An incision is then made in the outer layer of rubber to serve as the section, and the button is extracted as often as desired (figs. 8, 9, and 10).

These simple methods make it easy to practice. They require comparatively little time or effort and thus encourage regular daily practice, so essential to achieve a smooth technique for the intracapsular extraction.

129 Clinton Street.

THE AQUEOUS/PLASMA STEADY-STATE RATIO OF XYLOSE*

ITS COMPATIBILITY WITH THE SECRETION-DIFFUSION THEORY OF AQUEOUS-HUMOR DYNAMICS

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In a recent publication concerned with aqueous/plasma steady-state ratios of xylose and arabinose, Harris and Gehrsitz[1] presented experimental data which, they said, invalidates the mathematical representation of the concept of aqueous-humor dynamics proposed by Kinsey and co-workers (secretion-diffusion theory).[2-5] Harris and Gehrsitz reported that, in the rabbit, the steady-state ratio of these sugars varied significantly with the concentration in the plasma. That of xylose, for instance, increased from 0.25 to 0.65 with plasma concentrations of 2.0 and 5.0 mM/kg. of water, respectively.

In discussing their results, Harris and Gehrsitz pointed out that according to the concept of aqueous-humor dynamics of Kinsey and co-workers, the steady-state ratio of diffusible substances, such as sugars, depends on the relative diffusion rate of the compounds and the rate of flow of aqueous humor in a manner described by the following equation:

$$\frac{C_{aq}}{C_{pl}} = \frac{K_{diff}}{K_{diff} + K_{flow}}$$

where C_{aq} and C_{pl} represent the concentration in the aqueous humor and plasma, respectively, and K_{diff} and K_{flow} represent the coefficients of diffusion and flow, respectively.

Since, Harris and Gehrsitz continued, the diffusion rate would not be expected to vary with differences in the amount of material present in the plasma,† the observed variations in the steady-state ratio would have to result from alterations of the rate of flow (K_{flow}) if their data are to be explained on the basis of the secretion-diffusion theory. The flow rate, they said, would have to vary seventyfold to account for their data. They concluded their paper by saying that "A constant that could undergo such variations has no meaning. Thus their (Kinsey and co-workers') mathematical treatment and their conclusions drawn therefrom do not appear to be valid."

* From the Kresge Eye Institute.

† In the case of animals which were injected with gum arabic, the coefficient of diffusion probably increased significantly owing to the toxic effect of this substance on the iris or ciliary body.[6] Harris and Gehrsitz do not discuss the possibility that xylose might similarly alter one, or more, of the blood/aqueous barriers.

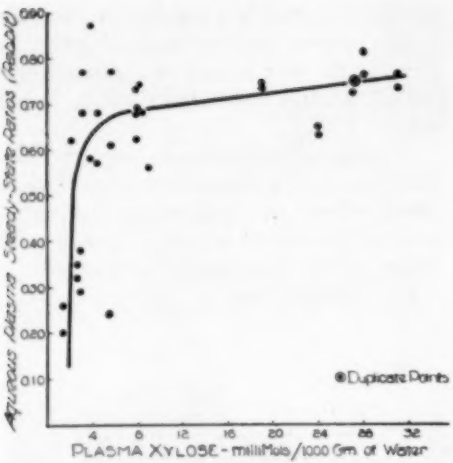

Fig. 1 (Kinsey and Frohman). A reproduction of Fig. 1 of the paper by Harris and Gehrsitz[1] (Am. J. Ophth., **34**: 113 [May, pt. 2] 1951). "Variation of the aqueous/plasma steady-state ratio with the xylose plasma concentration in the rabbit. Each point represents the value of one eye. A concentration of 32 mm./L. is the osmotic equivalent of 575 mg.-percent glucose."

The crux of the Harris and Gehrsitz argument seems to be that any mathematical equation involving a constant which must vary to represent the facts is both meaningless and invalid. With this argument there can be little disagreement, but it is not clear how this reasoning applies in the present instance since the coefficient of outflow, K_{flow}, was not conceived, or ever used, by the originators as a constant, but as a parameter.

A parameter, unlike a constant, may be defined as a coefficient which remains constant only as long as the limiting conditions under which it is evaluated do not vary; a constant, such as Π, h (Planck's constant), N (Avogadro's number), and so forth, is a number whose value never changes. The secretion-diffusion theory does not envisage that the coefficient of outflow must, or does, remain constant under any and all conditions.* Thus, to state that the theory is meaningless because the rate of flow of aqueous humor may not be constant reveals a fundamental misunderstanding of the nature of the theory.

The argument resolves itself, therefore, into the question of whether or not it is likely that the coefficient of outflow did vary sufficiently under the experimental conditions employed by Harris and Gehrsitz to account for the observed alterations in steady-state ratios of xylose.

Figure 1, which represents Harris and Gehrsitz's data for this sugar, shows that the major change in steady-state ratio of xylose occurred in the range of plasma concentrations of approximately 2.0 to 5.0 mM/kg. of water. The direction and magnitude of the change in coefficient of outflow necessary to account for their data can readily be calculated from the equation cited previously.

Using Harris and Gehrsitz's value for the turnover rate of xylose in the anterior chamber (2.84 percent per minute), which is numerically equal to $K_{diff} + K_{flow}$, the value for the flow rate is calculated to be approximately 5.5 percent per minute for a steady-state ratio of 0.25, corresponding to a plasma xylose concentration of approximately 2.0 mM/kg. of water, and approximately 1.0 percent per minute for a steady-state ratio of 0.65, corresponding to a plasma xylose concentration of approximately 5.0 mM/kg. of water.

The coefficient of outflow for the steady-state ratio of 0.70, corresponding to the maximum plasma xylose concentration reported, is approximately 7.0 percent per

* The most obvious instance of reduced flow of aqueous humor, of course, would be in a dead, or dying, animal. The literature offers various examples of conditions, both in experimental animals and in man, in which the coefficient of outflow probably was intermediate between that of a normal and dead animal. For instance, in cats, ether anesthesia, as Benham *et al*[7] have shown, raises the osmotic pressure of the plasma, thus altering the osmotic pressure relation between aqueous humor and plasma. Under this condition, the coefficient of outflow was no doubt reduced. In patients having glaucoma caused by blockage of the angle, the outflow of aqueous humor must also be reduced.

minute. The total variation in steady-state ratio is thus sevenfold.* The calculated values for K_{flow} are plotted as a function of plasma concentration of xylose and shown by the half-solid circles (joined by the solid line) in Figure 2.

If the line that represents the relation between steady-state ratio and plasma concentration of xylose (fig. 1) is extrapolated to include values for plasma concentrations less than 2.0 mM/kg. of water, it would pass through the base line representing zero steady-state ratio,† and would do so when the value of plasma concentration of xylose is still positive. It is evident from the equation that for any positive value of the coefficient of diffusion the flow rate would have to be infinite to account for a steady-state ratio of zero in the presence of a finite amount of xylose in the plasma. The flow rates calculated from values of the steady-state ratios extrapolated to 0.1 are indicated by the dashed-line extension of the solid line in Figure 2 .

A point representing the coefficient of flow determined experimentally in normal rabbits, that is, those which had not been injected with xylose,[4] is shown by the solid circle in Figure 2; the coefficients of outflow, calculated from data presented by Harris and Gehrsitz in a previous paper[8] concerned with the steady-state values and turnover rates for various sugars, including glucose, are shown by the open circles.

The solid line of Figure 2 indicates that the change in the coefficient of outflow necessary to account for the major change in the experimentally determined steady-state

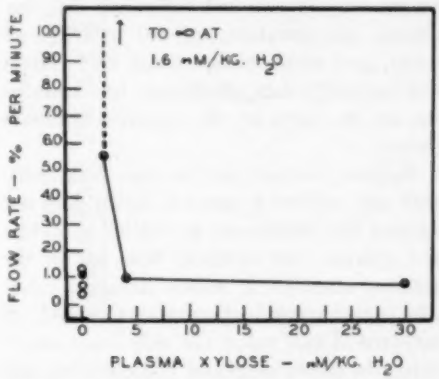

Fig. 2 (Kinsey and Frohman). Variations in the flow rate of aqueous humor which would be required to account for Harris and Gehrsitz's data relating steady-state ratio of xylose to plasma concentration.

values for xylose would be approximately five and one-half-fold.

While it is conceivable that a change in flow rate of this magnitude (that is, five and one-half-fold) might occur as a result of the injection of xylose into rabbits, the still higher flow rates required by plasma concentrations slightly less than 1.5 mM/kg. of water seem extremely improbable, particularly since evidence from other sources (represented by the solid and open circles) shows that the flow rate, far from being extremely high with zero plasma concentration of xylose, is about the same as that for a plasma xylose conceneration of 5 mM/kg. of water, and above.

Moreover, on the basis of purely pharmacologic considerations, it seems paradoxical that the range of plasma concentration in which the greatest changes in flow rate would have to occur would be that involving the lowest concentrations of xylose. Incidentally, the same reasoning applies to any explanation of why the steady-state ratio varied so widely with low, but not with high, concentrations of xylose in the plasma.

All of these considerations cast doubt on whether the flow rate ever, in fact, even approached the 5.5 percent per minute value

* Harris and Gehrsitz stated that the change in the coefficient of outflow necessary to account for their data would be seventyfold. This discrepancy apparently represents a miscalculation.

† The extrapolation apparently does no injustice to the data since in another curve shown by Harris and Gehrsitz which represents their data for arabinose, the steady-state ratio for the two lowest plasma values reported is actually zero, although they do not continue the line representing the data to include these points.

required by Harris and Gehrsitz's data for plasma concentrations of 5.0 mM/kg. of water, and make it improbable that Harris and Gehrsitz's data can actually be accounted for on the basis of the secretion-diffusion theory.

Neither, however, are the data compatible with any outflow hypothesis which does not assume that substances as similar as xylose and glucose, for instance, flow out of the anterior chamber at widely differing rates. The experimental observations would, if accepted at face value, not only force one to reject the theory originally proposed, but also any theory involving a nonselective outflow.

Since Harris and Gehrsitz themselves made no attempt to explain their data on the basis of any alternative mechanism, and since none of the various other possible hypotheses which occurred to the present authors (for instance, limited secretion out of the anterior chamber, limited hydrolysis in the anterior chamber, or variation of the rate of diffusion into the anterior chamber) seem very probable, particularly since these hypotheses in general could not account quantitatively for the entrance and exit of various other substances in the aqueous humor, as the secretion-diffusion theory does, the present authors were led to question the accuracy of Harris and Gehrsitz's experimental data.

An examination of these data (fig. 1) shows that the scatter of the points in the range of plasma concentration of 5.0 mM/kg. of water, and less, is so great that there is considerable doubt on statistical grounds whether Harris and Gehrsitz were justified in drawing any line through the points representing the steady-state values in this range of plasma concentrations.

Since these authors reported no data on the adequacy of their method of analysis, and since the method used is not specific for pentose but necessitates the removal of glucose by fermentation by yeast prior to performing the analyses, it is questionable whether it is adequate in the low range of xylose concentrations to which it was applied. These considerations led the present authors to repeat Harris and Gehrsitz's experiments, employing a method of analysis which was specific for pentose, and whose adequacy was established for the lowest range of xylose concentrations.

TABLE 1

ANALYSES OF XYLOSE SHOWING THE ACCURACY OF THE METHOD AND THE AMOUNT OF INTERFERENCE INTRODUCED BY VARIOUS COMPOUNDS

Quantity of Xylose Present (micrograms)	Quantity of Other Compound Present	Quantity of Xylose Recovered (micrograms)
5.0	—	5.3
10.0	—	10.2
15.0	—	15.0
20.0	—	19.8
20.0	100γ glucose	20.1
0.0	100γ glucose	0.2
20.0	100γ sucrose	19.9
0.0	100γ sucrose	0.0
15.0	25γ ascorbic acid	15.3
0.0	25γ ascorbic acid	0.4
20.0	100γ fructose	20.2
0.0	100γ fructose	0.2
20.0	100γ heparin	19.9
0.0	100γ heparin	0.0
20.0	2 drops 1% pontocaine	19.9
0.0	2 drops 1% pontocaine	0.0
20.0	0.1 ml. sat. sol. oxalate	19.8
0.0	0.1 ml. sat. sol. oxalate	0.0

EXPERIMENTAL

METHODS

Albino rabbits were used in all the experiments. Plasma levels of xylose were kept essentially constant by the following procedure. An injection of xylose was given intraperitoneally and followed 15 minutes later by an intravenous injection. Five additional intraperitoneal injections were then given at half-hour intervals. In the experiments in which the concentration of xylose in the plasma did not exceed 5.0 mM/kg. of water, the solution of xylose injected was isotonic with the plasma.

In order to obtain a plasma level of 1.0 mM/kg. of water, 18 mg. of xylose per kilogram of body weight were given intravenously and 20 mg. per kilogram of body weight were given in each intraperitoneal injection. Multiples of these quantities were

used to obtain higher plasma concentrations.

The concentration of xylose in the plasma was determined at 30-minute intervals to learn whether it remained essentially constant. In those instances in which the plasma level was not maintained almost constant the data were not included in the results. The concentration of xylose in a sample of aqueous humor taken two and a half hours after the first injection was determined and the steady-state ratios were calculated from this value and that for plasma obtained at the same time.

Blood was taken by heart puncture and aqueous humor by paracentesis under topical anesthesia. Heparin was used as an anticoagulant. No general anesthetic was employed.

The method of analysis for xylose was based on that of Mejbaum.[9] A reagent was freshly prepared by adding 10 mg. of orcinol to 10 ml. of 0.1-percent ferric chloride in concentrated hydrochloric acid. Three milliliters of this reagent were added to 3.0 ml. of a trichloracetic acid filtrate of the sample. The resultant solution was heated for eight minutes to develop a green color, and immediately cooled in an ice bath.

The amount of xylose present was determined from the absorption at 660 mμ in a Beckman spectrophotometer. Glucose, fructose, ascorbic acid, and sucrose were added to solutions containing known quantities of xylose in amounts as great as 500 μg. to determine whether these substances interfered with the method.

Results

The adequacy of the method of analysis and its specificity for xylose with respect to possible interference by other compounds are shown in the table. The method is accurate to better than two percent when used in determining quantities of xylose in excess of 10 μg. The minimum quantity of xylose determined in the biologic samples was 15 μg.; this sample was obtained from an eye of a rabbit whose plasma concentration was 1.4 mM/kg. of water.

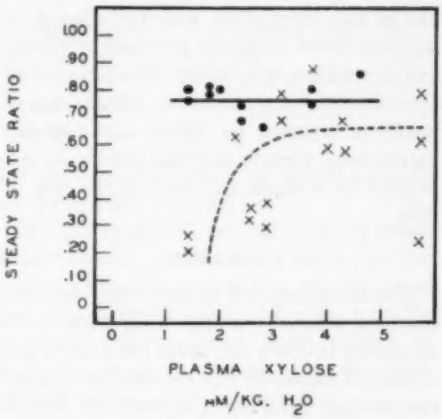

Fig. 3 (Kinsey and Frohman). Variation of the aqueous/plasma steady-state ratio with xylose plasma concentration in the rabbit. (Solid circles and solid line represent data obtained by present authors; crosses and broken line represent analogous data taken from paper by Harris and Gehrsitz.[1])

The ratio of the concentration of xylose in the aqueous humor to that in the plasma, plotted as a function of the plasma concentration, is shown by the solid circles in Figure 3. The crosses represent analogous data obtained by Harris and Gehrsitz, and the broken line is a reproduction (to scale) of the line they drew through their points. Only the lower range of plasma concentrations is included.

The results obtained by us (solid line) show that there is *no* significant variation in the steady-state ratio of xylose in the plasma range of concentration from 1.4 to 5.0 mM/kg. of water. These results are clearly different from those reported by Harris and Gehrsitz. The difference in the amount of scatter of the points in the two sets of data is apparent.

No alteration in flow rate need be assumed to account for the results obtained by the present authors; hence, the data substantiate the supposition based on purely logical considerations, cited earlier in this paper, namely, that the flow rate remains essentially constant. Furthermore, the data

are in fact compatible with the concept of aqueous humor dynamics proposed by Kinsey and co-workers. The steady-state ratios in the concentration range of above 5.0 mM/kg. of water as reported by Harris and Gehrsitz varied only slightly and can readily be explained by a slight variation in the rate of flow.

Summary

The reasoning and experimental observations on aqueous/plasma steady-state ratios of xylose that are the basis for Harris and Gehrsitz's statement that the concept of aqueous humor dynamics proposed by Kinsey and co-workers is invalid are critically examined. Harris and Gehrsitz's logic is shown to be based on a misconception of the nature of the coefficient of outflow and their experimental data are shown to be questionable.

We repeated the experiments performed on rabbits by Harris and Gehrsitz (but without general anesthesia) using a method which was specific for pentoses and whose adequacy in the low range of plasma concentration was demonstrated. The results obtained differ from those of Harris and Gehrsitz and show that the steady-state ratio of xylose did not vary appreciably with the concentration of this sugar in the plasma in the range of concentration between 1.4 and 5.0 mM/kg. of water. These results, which give rise to flow rates of aqueous humor comparable with those obtained by various other procedures, agree with values predicted on the basis of the theory of aqueous humor dynamics (secretion-diffusion theory) as previously outlined, and to this extent substantiate the validity of the theory.

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Acknowledgment

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LATE CHANGES IN THE VITREOUS FOLLOWING UNCOMPLICATED INTRACAPSULAR CATARACT EXTRACTION*

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When the intracapsular method of cataract extraction was first advocated there were dire predictions that the resulting exposure of the vitreous to the action of the aqueous would produce degenerative changes within the eye which would negate the initially good visual results. It was further stated that the loss of support of the lens would result in vitreous displacement with consequent secondary glaucoma, retinal detachment, and the like. As improved surgical techniques made the operation available to large numbers of surgeons, it was seen that these predictions were largely unfounded.

In 1947, Knapp[1] stated: "It has been claimed that the intracapsular operation unduly traumatizes the eye, displaces the pupil, and causes degenerative changes in the vitreous which damage the eye."

He reëxamined 57 patients who had been operated upon 10 to 15 years before and found that vision was as good as immediately after the operation. Furthermore, there were no changes demonstrable in the vitreous of these cases which could be attributed to delayed degenerations. If the primary result was good, excellent vision was retained.

Knapp further stated that: "The importance of the zonulo-capsular barrier for the integrity of the eye is an unsettled question and one that requires further study. The resulting displacement and the disturbed relationship of important structures in the interior of the eye suggest the possibility of damage to the eye, but late examination of many patients and clinical experience have not shown any evidence of deterioration of the eye from this cause."

Wright[2] felt that an eye with an intact zonulo-capsular barrier was a better eye than one in which this barrier was not intact and that the integrity of the vitreous must be preserved. These views were shared by numerous surgeons, especially in the early days of the intracapsular operation.

In later years, a number of authors, perhaps puzzled by the absence of expected deterioration in eyes operated upon by the intracapsular method, have reëxamined cases to determine what changes, if any, occurred in the bared vitreous after an uncomplicated intracapsular cataract extraction. Their findings have varied.

Elschnig[3] found the vitreous protruding through the pupil and into the anterior chamber and covered by a membrane riddled by many small holes through which free floccules of vitreous extended, often as far forward as the cornea.

Cowan and McDonald[4] found that a bulging vitreous flattened out, and the hyaloid membrane did not thicken. If a tear developed, the vitreous extended forward to the cornea without damage. They stated that the squeezing of the bulging vitreous by the contracted pupil might cause glaucoma, and this statement has been echoed by Knapp[1] and Pereira.[5]

In Kirby's[6] experience the protrusion of the vitreous gel into the aqueous was rather rare and not productive of further pathologic disturbances. He also felt that the round pupil held back the vitreous better than when a coloboma was present, and that there was, in the average case, less vitreous in the anterior chamber in the presence of a round pupil than with a complete coloboma.

Reese[7] and Leahey[8] have described at

* From the Division of Ophthalmology, University of California Medical School, and the Veterans Administration Hospital, Fort Miley, San Francisco. The drawings are by Kay Hyde, U. S. Veterans Administration Hospital, San Francisco. Read at the meeting of the American College of Surgeons, San Francisco, November 8, 1951.

length the bullous keratitis resulting from vitreous contact with the posterior surface of the cornea. Both felt that it was a serious complication.

Leahey estimated that some permanent vitreous contact was present in over 10 percent of intracapsular cataract extractions but that areas of resultant corneal edema occurred in only 0.5 percent of aphakic eyes. All of his cases had full iridectomy and, in over half of the cases showing vitreous contact, it occurred as a peripheral one at the top of the complete iridectomy area. He stated that the factors producing corneal edema were as follows: (1) Duration of contact; (2) density of contact; (3) location of contact; (4) condition of the endothelium at the time of contact; (5) unknown factors, and outlined an effective method of treatment for the condition.

In a series of 522 cases, Kubik[9] concluded that vitreous herniation was largely temporary and devoid of any late ill effects. He found no cases of keratitis from vitreous contact.

It is obvious that certain late changes do occur in the vitreous following uncomplicated intracapsular cataract extraction. What are these changes? What is the importance of the zonulo-capsular barrier? Which of these late vitreous changes are harmful and which inocuous? What can be done to minimize the occurrence and effect of those changes which might be harmful?

In reëxamining and analyzing a series of 66 selected eyes which had had uncomplicated intracapsular cataract extractions, I did so with the intent of studying: (1) The types of vitreous changes which occurred; (2) the effect of these changes on the integrity of the eye and, more important, on the function of the eye; (3) the manner in which these changes might indirectly produce secondary serious ocular pathology; (4) the effect of certain surgical techniques in the production of late vitreous changes; (5) the possibility that some of these changes in the vitreous might have preëxisted the cataract extraction; (6) techniques which might be employed to decrease the incidence of the possibly harmful vitreous change.

It is not the intent of this paper to analyze statistically a large series of cases of intracapsular cataract extractions. This has been done by the authors already quoted. The cases upon which this study is based have been selected (1) to provide a cross-section of the various details of technique in lens extraction; (2) because in all there was a visual acuity of 20/20 or better when first tested postoperatively; (3) because they covered a postoperative period of from nine days to 11 years; (4) because no case exhibited any complication of any kind at operation, or postoperatively, as a result of the surgery.

With these criteria in mind certain statistics regarding these cases are of interest:

1. The average interval between operation and examination was two years and three months; with the shortest period of nine days, and the longest, 11 years.

2. Sixty lenses were extracted through a round pupil with peripheral iridotomy.

3. Six cases had a full iridectomy.

4. In 59 cases the lens was tumbled after dislocation.

5. In seven cases the lens was slid from the hyaloid fossa head first.

6. Thirty-six lenses were extracted with the Harrington[10] erisophake.

7. Thirty lenses were extracted by the method of external pressure and forceps traction.

8. Fifty-two eyes had an air bubble injected into the anterior chamber at the conclusion of the operation.

The following postoperative observations were made upon the vitreous and will be discussed in some detail.

1. The body of the vitreous appeared viscid in 57 cases and fluid in nine.

2. The anterior face of the vitreous (hyaloid) was flat in 25 cases; was simply and

moderately herniated in 25 cases; was markedly herniated in mushroomlike shape in 12 cases; and was apparently totally absent in four cases.

3. Of 40 eyes specifically examined for this defect, eight showed a hole in the posterior hyaloid with posterior vitreous detachment.

4. Nine cases showed a single hole in an otherwise intact anterior vitreous face.

5. Four eyes showed two or more holes in an otherwise intact hyaloid.

6. One case developed a persistent contact of the vitreous face with the corneal endothelium.

7. Two eyes showed slight thickening of the anterior vitreous face.

8. Four eyes showed reduction in vision below the immediate postoperative visual acuity of 20/20 or better, from the following causes:

a. Slowly progressive disciform degeneration of the macula.
b. Acute central choroiditis, unrelated to surgery and appearing three years postoperatively.
c. Corneal scar resulting from ulceration of cornea two years after operation.
d. Progressive postoperative thickening of the anterior hyaloid.

From these cases, plus a study of case reports from the literature, plus personal communication with other observers, certain valid conclusions may be drawn regarding each of the late changes in the vitreous just noted.

In the majority of operations through a round pupil there is an immediate postoperative herniation of the vitreous, of moderate degree, which shows pigment deposits on its face, which is smooth, which is not much affected by the action of the pupil, and which gradually flattens until it is in the plane of or behind the iris. The longer the interval of time between operation and observation the higher the percentage of flat hyaloid membranes. There were a few cases in which the herniation showed no change whatever after several years. Holes in the vitreous face were seen in both flat and herniated cases.

The herniation of the vitreous extended farther forward into the anterior chamber in cases with full iridectomy, although in some of the round-pupil extractions with marked herniation through a small pupil, the vitreous appeared to extend far forward because of its mushroomlike appearance.

The only case in this series which showed persistent contact between vitreous face and posterior corneal surface was one with a full iridectomy. This is in agreement with Leahey's[8] experience. In no case were there any corneal changes or visual loss.

In the 12 cases which showed an extreme degree of mushroomlike herniation of vitreous through a small round pupil, not one gave evidence of the so-called pupillary block glaucoma or even any significant variation in intraocular pressure. It was especially noted that in these cases a change in pupillary size did not alter the degree of vitreous protrusion, an indication of freedom from adhesion between vitreous face and iris.

In one case of this type (fig. 1), a maxi-

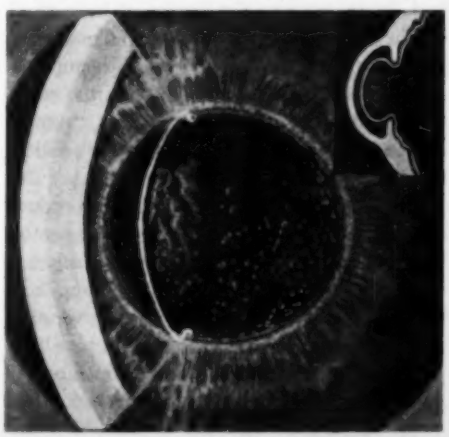

Fig. 1 (Harrington). Simple vitreous herniation of considerable degree. Hyaloid membrane intact with considerable pigment deposit.

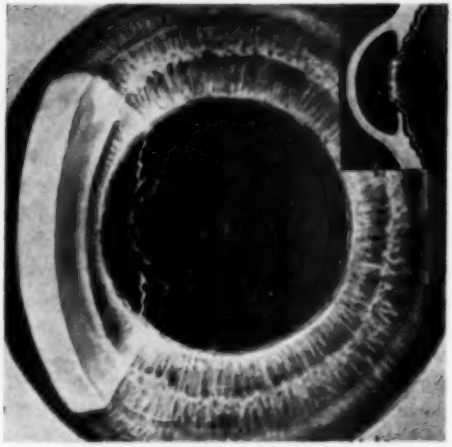

Fig. 2 (Harrington). Large central hole in hyaloid membrane giving the effect of an absence of vitreous face.

mum herniation of vitreous was allowed through wide dilation of the pupil after which the pupil was sharply contracted behind the protruding hernia, pinching it off like a mushroom. There was no rise in intraocular pressure even though there was an apparent complete pupillary block.

It is my belief that pupillary-block glaucoma does not occur unless there is an annular posterior synechia between iris and hyaloid. In these cases the degree of herniation of the vitreous varies with the size of the pupil, being pulled flat when the pupil is dilated and moving forward when the pupil is contracted. Also, in these cases there is evidence of postoperative iridocyclitis and in the later stages a true iris bombé may develop. In one such case I was able to reduce intraocular pressure, apparently permanently, by dilating the pupil sufficiently to produce a tear in the hyaloid membrane at the pupillary border. Vitreous herniated freely through this tear but subsequent changes in pupillary size did not produce a rise in intraocular pressure.

The degree of vitreous herniation is apparently not influenced by the method of lens extraction. It is about the same in those cases where lenses were dislocated and delivered by the method of external pressure and forceps traction, where the ratio of pressure to traction is approximately 5:1, and those in which the erisophake was used where the ratio of pressure to traction might be 1:5. It also does not seem to be influenced by the tumbling or sliding method of delivery. Nor does damage to the face of the vitreous appear to be greater in one procedure than the other.

If vitreous can be prevented from immediate postoperative contact with an injured corneal endothelium, it would appear that persistent vitreous-corneal contact with bullous keratitis, as described by Reese[7] and Leahey,[8] could be largely avoided. This can usually be accomplished by the injection of a small amount of air into the anterior chamber at the close of the operation.

I have not succeeded in duplicating Scheie's[11] observations of a rise of intraocular pressure following air injected into the anterior chamber. I do not believe that "pupillary-block glaucoma" is produced by the blockage of the anterior-posterior chamber communication by a bubble of air unless, perhaps, it would be by a very large bubble which was incapable of moving in the pupillary arc with movement of the head and took a number of days to absorb. A small bubble of air in the anterior chamber will effectively hold the vitreous back and, at the same time, will cause only very slight and intermittent block in the pupil, easily bypassed by increased aqueous pressure.

If the head is elevated or turned slightly from one side to the other from time to time, the air bubble can be seen to move from its central position in the pupil just as does the bubble in a spirit level. Each time the bubble moves, even though very slightly, the anterior chamber to posterior chamber communication can be seen to open. With a small air bubble the normal amount of freedom of movement allowed the cataract patient is sufficient to accomplish this. At the same

time, there is little or no chance for forward displacement or herniation of vitreous until the bubble is completely absorbed, after which the major complication of vitreous-corneal contact is unlikely.

A larger bubble of air is required to hold back the vitreous effectively in the presence of a full iridectomy than in a round-pupil extraction. This is especially true in the area of the incision where it is most important that no contact between vitreous face and damaged corneal endothelium be allowed.

Cases are mentioned in the literature in which the vitreous seems to spill forward into the anterior chamber without form or barrier. In this series there were four such cases. One of these (fig. 2), after dilation of the pupil, proved to have a very large centrally placed hole in the hyaloid through which a semiviscid vitreous protruded into the anterior chamber. In the other three cases no sign of a hyaloid membrane or vitreous face could be demonstrated and a semifluid mass appeared to pour forth through the pupil, spilling over its borders and filling the anterior chamber. In no case, after two, four, and six years respectively, had this phenomenon produced any harmful effect upon the eye, either as to its integrity or function. In all cases the vitreous was crystal clear to ophthalmoscopic examination and vision was 20/20 or better.

The presence of a small hole in the vitreous face after uncomplicated intracapsular cataract extraction has been mentioned by many authors[1, 3, 4–6, 9] and there were nine such cases in this series (fig. 3). Such single holes in the hyaloid were seen after intervals of from five months to 11 years. All but one were in eyes examined after intervals of four years or more.

The holes may appear at any point in the hyaloid, but the majority were at the pupillary border, suggesting that they were tears resulting from the action on the vitreous face of an iris-vitreous adhesion. In each case the edge of the hole looked slightly thicker

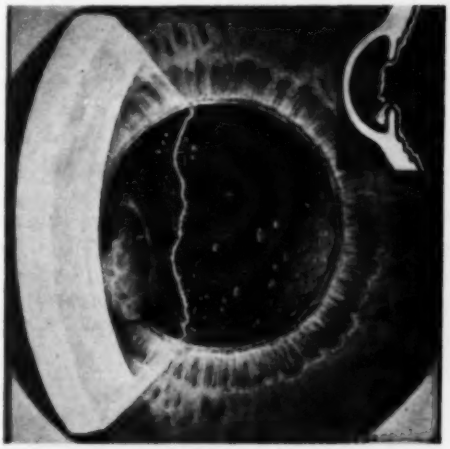

Fig. 3 (Harrington). Single hole in vitreous face at pupillary border. Note flaccid, nonherniated hyaloid.

than the surrounding hyaloid and a small mass of viscid or semiviscid vitreous protruded a short distance into the aqueous.

In several instances, these holes were extremely difficult to see even under the high power of the slitlamp. In a few instances the faint outline of the hole could be detected with the +16.0, D. lens of the ophthalmoscope. In no case was there any apparent deleterious effect upon the eye or its function.

Irvine,[12] in a personal communication, has mentioned that in some of his postoperative examinations of intracapsular cataract extractions, the vitreous face gave a sieve or lacelike appearance. There were many tiny holes, each with its bead of vitreous extruded into the anterior chamber. He was much concerned as to the possible late effect of these changes on the visual function. Elschnig[3] had made note of these same findings.

In this series, there were four such cases with multiple holes in the vitreous face (fig. 4), a lesser percentage than that found by other authors. One eye showed four small holes in the vitreous face, while the other three eyes showed two holes. In all cases the vitreous was only very slightly herniated

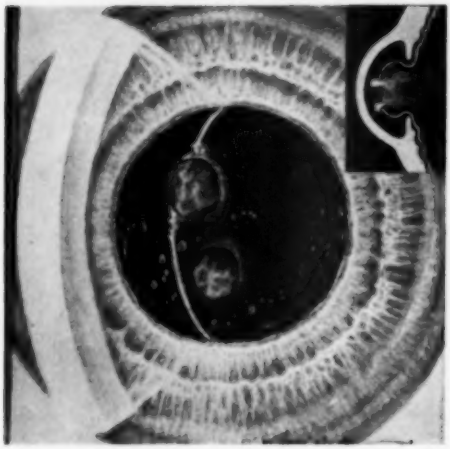

Fig. 4 (Harrington). Moderate anterior vitreous herniation with two holes in the hyaloid.

through the pupil and only thin strands of loose vitreous were seen to project through the holes into the aqueous. The vitreous had remained clear and vision was unaffected.

Thickening of the face of the vitreous or hyaloid membrane has not been reported in the studies dealing with examinations of the vitreous after uncomplicated cataract extraction except as it occurred as a result of vitreous-corneal contact or in postoperative uveitis with cyclitic membrane formation.

In a personal communication, Dr. John Dunnington[13] reported one case of unknown etiology in which a diffuse thickening of the hyaloid membrane occurred sufficient to reduce visual acuity materially.

Dr. Howard Mallek,[14] of Vancouver, British Columbia, told me of two eyes in one patient in which uncomplicated intracapsular cataract extractions were followed by gradual thickening of the vitreous face sufficient to reduce vision markedly and require bilateral discissions.

Dr. F. A. Davis[15] reported that he had never seen a thickening of the hyaloid in an uncomplicated intracapsular extraction although he had seen one or two which followed persistent recurrent hyphema or postoperative uveitis. He stated that late rupture or tear of hyaloid was commonly seen but was not followed by any complication. It was his observation that adhesion of the hyaloid to the posterior surface of the cornea occurred immediately after extraction and was not a late complication.

In this series of 66 cases, there were two eyes which developed detectable thickening and opacification of the hyaloid. In one eye (fig. 5) the membrane slowly developed a diffuse, mottled, faint gray clouding which appeared to be due to irregular thickening of the vitreous face. The opacity was first noted about one month postoperatively and progressed very slowly during the next year until vision was reduced from 20/20 to 20/40, after which, during a three-year interval, no further changes took place.

Another eye showed a faint linear streak across the hyaloid which was seen to be a definite thickening of the membrane. It was first noted about three weeks postoperatively and has remained completely unchanged during the past year and a half. Vision has remained unaffected at 20/20. It would seem that this phenomenon, while very uncom-

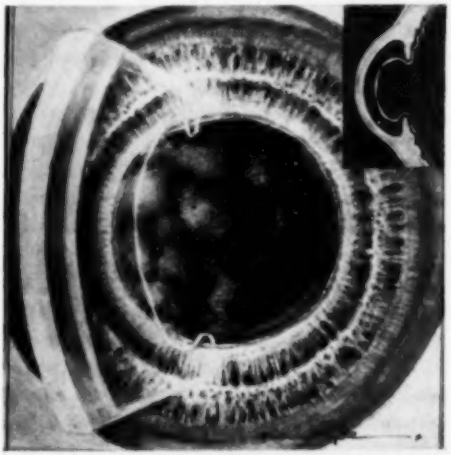

Fig. 5 (Harrington). Diffuse patchy thickening and opacification of the vitreous face.

mon, is one which might be more often seen if searched for, and which must be reckoned with as a possible cause of visual loss after baring of the vitreous.

There has been much discussion of the danger of retinal detachment following intracapsular cataract extraction. It is now fairly generally agreed that the incidence of retinal detachment is about the same after both intracapsular and extracapsular cataract extraction. It is also rather widely accepted that detachment of the posterior vitreous is a determining factor in the production of retinal detachment.

Of 40 eyes in this series specifically examined by slitlamp and Hruby or contact lens for posterior vitreous changes, eight were found to have a greater or lesser degree of posterior vitreous detachment with a hole in the posterior hyaloid. This 20-percent incidence is considerably less than might be expected. Dr. Dohrmann Pischel[16] has estimated that "more than 40 percent of normal eyes in the cataract age group have posterior vitreous detachment." Certainly this series, small though it is, would not bear out the theory that destruction of the zonulocapsular barrier contributes to posterior vitreous detachment and secondarily to retinal detachment.

Summary and Conclusions

The late changes in the vitreous following uncomplicated intracapsular cataract extraction may be listed as follows:

1. Simple herniation of the vitreous face through the pupil.
2. Herniation of the vitreous face with potential pupillary-block glaucoma from vitreous-iris adhesion.
3. Herniation of vitreous with persistent vitreous-corneal contact and the rare occurrence of bullous keratitis.
4. Absence of the vitreous face.
5. Single or multiple holes in the anterior hyaloid membrane.
6. Thickening and opacification of the anterior vitreous face.
7. Posterior vitreous detachment.

From a review of the literature and an analysis of 66 selected cases the following conclusions may be drawn:

1. The zonulo-capsular barrier is of little or no importance as far as the integrity and visual function of the eye is concerned.
2. With three possible exceptions, the late changes in the vitreous following uncomplicated intracapsular cataract extraction have no adverse effect upon the eye or its function.
3. Persistent contact between the exposed vitreous and an injured cornea may give rise to bullous keratitis and visual loss.
4. A rare thickening and late opacification of the anterior vitreous face of unknown etiology may cause some loss of visual acuity.
5. The rare occurrence of complete annular synechia between vitreous face and iris may give rise to the so-called "pupillary-block glaucoma."
6. Vitreous-corneal contact is less prevalent in round-pupil extractions than with full iridectomy, and its incidence may be decreased by the routine injection of a small air bubble into the anterior chamber at the close of the operation.
7. Incipient iris-vitreous adhesion may be detected by observation of the pupillary action on the degree of vitreous herniation, and prevented by pupillary dilation. It is not likely to occur in the absence of postoperative inflammatory reaction in the aqueous.
8. No special method of lens extraction can be said to influence the late changes in the vitreous.
9. The incidence of posterior vitreous detachment is no greater after uncomplicated intracapsular cataract extraction than in normal eyes in the same age group.
10. The degree of fluidity of the vitreous does not appear to affect the late changes.

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STREPTOKINASE IN OPHTHALMOLOGY

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Streptokinase (streptococcal fibrinolysin) is a natural product of certain Lancefield groups of hemolytic streptococcus. It acts indirectly on a substrate of fibrin or fibrinogen by activating a fibrinolytic enzyme in human serum which then splits fibrin into polypeptides, and thus causes rapid dissolution of blood clots and fibrinous exudates.

Streptokinase is, then, a catalyst which aids in the transformation of a zymogen in human blood into a proteolytic enzyme. This enzymatic effect begins immediately upon application at the site of the disease process, progresses for approximately 24 hours, and is generally self-terminating. The amount of human serum available limits the activity.

Clinical need for a material which will bring about dissolution of blood clots is abundant. Streptokinase has been tried in the following conditions, in many of which a blood clot or fibrinous exudate was a major factor: hemothorax and hematoma, suppurating disease such as osteomyelitis, otitis media, burn infections, and pneumonitis.

Reports state that great amounts of streptokinase may be injected intrathecally without danger. When used in the thoracic cage, a local irritation is set up which results in the outpouring of red and white blood cells along with serum. Local irritation ceases in one to two days, only rarely results in a pleural adhesion.

It is thought best not to use streptokinase in the face of active bleeding or acute nonsuppurative cellulitis. Other reactions to the use of the drug have paralleled those to any parenterally administered foreign protein. Fever to 103°F., chills, headache, nausea, vomiting, general malaise, and arthralgia may be encountered.

Ophthalmologists have long been perplexed by the problem of hyphema. Not only is the original hemorrhage into the anterior chamber a serious condition, but even more disturbing is the secondary or "delayed" hemorrhage. This secondary hemorrhage is of greater intensity than the original hyphema, and generally occurs on the third to

fifth posttraumatic day. Increased intraocular pressure frequently accompanies either the primary or delayed bleeding. Subsequent complications may include blood-staining of the cornea, atrophy of the iris, secondary membrane formation, and glaucomatous optic atrophy.

Jukofsky recently reported a case of traumatic primary and secondary hyphema which cleared when treated with streptokinase injected into the anterior chamber. He also reported a case of subconjunctival hemorrhage which cleared upon injection of streptokinase subconjunctivally. It would certainly seem that streptokinase offers a simple agent with which to treat a very serious condition and, perhaps, thereby avoid the complications usually encountered.

Case reports

Case 1

L. D., a nine-year-old white boy, was admitted to the ophthalmology service of the Los Angeles County General Hospital four days after having been struck in the left eye with the butt of a toy revolver.

On admission his vision was: R.E., 20/15; L.E., light perception. The trauma was limited to the left eye which showed the anterior chamber to be completely filled with dark blood. The iris details were completely obscured. The patient had had an original and immediate small hyphema, but it was severe secondary hemorrhage which brought the child to the hospital.

The tension in the injured eye was 52 mm. Hg (Schiøtz). After intensive miotic therapy had failed to reduce the tension, a paracentesis incision was made at the limbus at the three-o'clock position with a beveled von Graefe knife. After inserting a 26-gauge needle through the opening into the anterior chamber, 0.2 cc. of very bloody fluid was removed. A solution of equal volume containing 50,000 units of streptokinase in saline was injected into the anterior chamber with the same needle.

In 12 hours, the iris outline could barely be seen. In 24 hours, the blood was almost gone, but in its wake was left a severely irritated eye with a blood-stained cornea, marked ciliary injection, corneal edema, and conjunctival chemosis. The irritation has persisted, and the eye has gone on to develop a dense cyclitic membrane, permanent corneal edema, and small endothial opacities. The tension is now consistently soft. Vision remains at light perception only.

Case 2

S. A., a 24-year-old woman, was admitted to the ophthalmology service of the Los Angeles County General Hospital for treatment of a severe traumatic subconjunctival hemorrhage of the right eye.

Visual acuity was normal in each eye and there were no pathologic findings in the posterior segments. A subconjunctival injection of a saline solution of 50,000 units of streptokinase was made in the area of the greatest hemorrhage.

In one-half hour there was a massive chemosis which was accompanied by corneal edema, severe burning pain, marked lacrimation, and photophobia. For the next three hours the condition became worse in spite of ice packs and adrenalin instillations. By the next morning the cornea was clear, but the chemosis and pain did not disappear for 72 hours. The hemorrhage itself did not resolve for about 10 days.

Case 3

C. J., a 58-year-old Negress, was admitted to the ophthalmology service of the Los Angeles County General Hospital for the treatment of secondary (aphakic) glaucoma of her right eye.

On admission her vision was: R.E., 20/30; L.E., 20/30 with her cataract correction. A cyclodialysis was performed on the right eye without incident.

Three days after surgery, while reaching for her water bottle, the patient experienced a sudden pain in the operated eye. Examination revealed the anterior chamber to be completely filled with blood. In the course of the next two days the blood became dark and the intraocular pressure rose rapidly.

Since the blood had clotted and the tension was climbing, 35,000 units of streptokinase were injected into the anterior chamber in the manner identical to that described in Case 1. In 24 hours, the iris structures could be seen, and the tension began to fall, but there developed an endothelial haze which has persisted. Severe irritation, cyclitis, and formation of a cyclitic membrane occurred. The vision dropped to perception of light in the involved eye. Moderate injection, tenderness, and occasional pain continue.

Case 4

M. M., an 11-year-old white boy, was admitted to the ophthalmology service of the Los Angeles County General Hospital after being struck on the right eye by a "BB."

On admission, he presented a small laked hyphema in the right eye that did not cover the pupil or obscure the vision. The tension was soft and there was no corneal edema or haze. X-ray as well as clinical examination showed no retained intraocular foreign body. Both eyes were patched, the child was sedated and placed on strict bedrest.

Four days after admission, a secondary or de-

layed hyphema occurred which completely filled the anterior chamber and obscured all the iris detail. The tension rapidly climbed to 47 mm. Hg (Schiøtz). In two days the blood in the anterior chamber showed no signs of absorbing. The tension remained elevated and the cornea became blood-stained.

An injection of 25,000 units of streptokinase was made into the anterior chamber in the manner described in Case 1. Twenty-four hours later the eye became painful for the first time. There was marked injection, deep corneal haze, conjunctival chemosis, and epiphora. The blood was gone from the anterior chamber in 36 hours but a cyclitic membrane rapidly took its place. In spite of all therapy, light perception was lost. Three months after the injury, the right eye was enucleated because of pain.

Case 5

S. B., a 19-year-old Negro, was admitted to the ophthalmology service of the Los Angeles County General Hospital for the treatment of a severe subconjunctival hemorrhage as a result of a blow to the left eye.

Vision was normal in both eyes and there were no findings other than the subconjunctival hemorrhage in the left eye. Streptokinase (25,000 units) in a saline solution was injected into the area of the greatest bleeding. There followed a rapid development of pain, tenderness, tearing, photophobia, and massive chemosis. The pain was relieved in 48 hours by cold compresses and adrenalin instillations, but the chemosis persisted for four days. The blood under the conjunctiva cleared in nine days.

Discussion

The treatment of hyphema by the use of direct intraocular injection of streptokinase was attempted in three different eyes. In all three cases the result was bad. Two attempts were made to treat subconjunctival hemorrhage with local injection of streptokinase. These two cases were also failures. What information can we gather from these failures?

It may be said to the drug's credit that the blood in the anterior chamber actually did disappear much more rapidly after injection than if left to absorb naturally. In all cases, the iris shadow could be made out in about 12 hours, the iris detail in about 24 hours, and the blood was almost completely gone in about 36 hours. The subconjunctival blood did not disappear so rapidly.

In all instances, the reaction to the irritation of the streptokinase was almost worse than the original condition for which it was used. In each case of hyphema, the endothelial damage with subsequent corneal edema, the severe cyclitis and secondary cyclitic membrane, and the chronic inflammation were collectively responsible for loss of useful vision. In one case, the resultant secondary glaucoma and pain were severe enough to require enucleation.

It is impossible to predict what the end result of these cases might have been without streptokinase treatment.

In the cases of subconjunctival hemorrhages, the chemosis, lacrimation, photophobia, and pain which occurred following the injection of streptokinase were far worse than a large asymptomatic subconjunctival hemorrhage.

The dosage at first paralleled that suggested in Dr. Jukofsky's article. The method of preparation and injection were similar. Cutting the dosage in half did not seem to remove the markedly irritating qualities of the drug.

Summary

Contrary to the original hope, the use of streptokinase in its present form is not the answer to the great problem of intraocular hemorrhage. The drug is now considered to be too irritating for injection into the human eye.

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AUREOMYCIN IN TRACHOMA*

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Since aureomycin has a specific activity against viruses of the lymphogranuloma-inguinale/psittacosis group, it seems reasonable, on theoretical grounds, to expect it to constitute a specific therapeutic agent against trachoma. Up to date, however, reports of practical application of the drug have been scanty.

Braley and Sanders,[1,2] Bellows and others,[3] Moutinho,[4] Lopes d'Andrade and Ribeiro Breda,[5] and especially Boase[6,7] from Uganda have reported very encouraging results from the use of aureomycin in cases of trachoma. This early optimism strongly reminds us of the exaggerated hopes which the sulfonamides aroused some 10 years ago with regard to their specificity against trachoma.

The aim of this article is to publish the results of trials carried out on 20 trachoma patients treated with aureomycin. The number is very small because of the limited quantity of aureomycin placed at our disposal. Nevertheless, the uniformity of our results makes us feel that they are highly significant.

As far as we know, this is the first report denying the effectiveness of aureomycin against trachoma. It is very likely that it may start a controversy which, as in the case of the sulfonamides, may continue until the efficacy of aureomycin has been established or disproved, or until a newer drug usurps our attention and make further discord unnecessary.

CHOICE OF CASES

To avoid wrong diagnosis, simplify dosage, and facilitate conclusions, a strict standard of choice was maintained throughout the series. This was not difficult on account of the great number and variety of trachoma patients attending our clinics. All patients were children four to eight years of age who had had no previous treatment.

All cases had typical trachoma II (MacCallan) with an admixture of follicles and sago grains (bleblike excrescences which burst on pressure) and a pannus at least four to five mm. wide. We chose this type because it is the most frequently met with in our clinics and also because diagnosis in these cases can be made with great ease and certainty. Thus no doubtful cases were included. Inclusion bodies were not looked for as in Lebanon this has only an academic interest.

As most workers on trachoma assess their results of treatment on the subjective improvement that the patients report, we tried to include in our series only those patients

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TABLE 1

CASES TREATED WITH 0.5-PERCENT AUREOMYCIN DROPS
(Number of pluses indicates severity)

Case No.	Sex	Age	Follicles	Excrescences	Pannus	Culture		Result
						Before Treatment	After Treatment	
1	M	8	+	+++	++	Streptococcus viridans	No growth	No change
2	F	7	+	+++	O.D. + O.S. +++	Koch-Week's bacillus	No growth	No change
3	M	5	++	+++	+++	Koch-Week's bacillus	No growth	No change
4	F	8	+	++	++	Staphylococcus albus	No growth	No change
5	M	4	+	+++	++	Streptococcus viridans	No growth	No change
6	M	4	++	++	++	Staphylococcus albus Diphtheroids	No growth	No change
7	F	5	+	++	+	Streptococcus anhemolyticus	No growth	No change
8	F	6	++	+	+++	Streptococcus hemolyticus	No growth	No change
9	F	8	+	++	+++	Pneumococcus	No growth	No change
10	F	8	+	++	++	No growth	No growth	No change

TABLE 2

CASES TREATED WITH LOCAL AUREOMYCIN OINTMENT AND CAPSULES PER OS
(Number of pluses indicates severity)

Case No.	Sex	Age	Follicles	Excrescences	Pannus	Culture		Result
						Before Treatment	After Treatment	
1	M	5	+	++	++	Koch-Week's bacilli	No growth	No change
2	M	5	++	++	++	Pneumococcus	No growth	No change
3	M	4	++	++	++	Streptococcus viridans	No growth	No change
4	F	7	+	+	+++	Staphylococcus albus	No growth	No change
5	M	4	++	++	++	Bacillus subtilis	No growth	No change
6	M	8	+	+	+++	Bacillus pyocyaneus	No growth	No change
7	F	5	++	+++	++	Morax-Axenfeld bacilli	No growth	No change
8	F	6	+	++	++	Pneumococci	No growth	No change
9	M	4	++	++	++	No growth	No growth	No change
10	F	6	++	++	+++	Staphylococcus aureus	No growth	No change

who had minimal or no symptoms whatsoever. As a result many of the patients who were chosen for this study had originally presented themselves for treatment of conditions other than trachoma (squint, error of refraction, otitis media, hypertrophied adenoids, and so forth).

The bacteriologic flora of the conjunctiva was studied before and again 48 hours after commencement of therapy.

All patients were hospitalized for the duration of the treatment.

Methods of treatment

The first series (table 1) consisting of 10 patients were treated with hourly instillations of 0.5-percent aureomycin borate solution into the conjunctival sac for a period of 12 days (12 instillations per day).

The second series (table 2) consisting of 10 other patients received the following joint treatment: (1) Local application of aureomycin hydrochloride ointment (1.0 mg. per gm. of base) every two hours for 12 days, together with (2) one aureomycin hydrochloride capsule (250 mg.) every six hours (1.0 gm. per day) for 12 days.

Criteria of cure

A recognized, standard method of assessing improvement in cases of trachoma undergoing treatment has not been formulated.

The advent of the sulfonamides, aureomycin, chloromycetin, and probably other future antibiotics necessitates the provision of an international standard by which the efficacy of the various drugs may be estimated.

The following, we believe, should be considered in assessing improvement or cure in cases of early trachoma:

1. The number of follicles.
2. The density and size of sago grains (MacCallan's bleblike excrescences).
3. The width and thickness of the corneal pannus.

In our opinion, conjunctival papillae and the subjective symptoms of the patient should not be taken into consideration as both are mainly due to secondary bacterial infection and are not dependent on the trachomatous process itself.

Results

Every case was studied by slitlamp before the commencement of treatment and again after the completion of the course of therapy.

In both series of patients our results were essentially similar.

After 12 days of treatment, the follicles, the bleblike excrescences, and the corneal pannus were essentially unchanged.

Since our patients had minimal or no subjective symptoms to begin with (blepharospasm, photophobia, lacrimation, itching, or discharge) they felt no change at the end of the course and actually expressed the desire to go home "as we were doing nothing for them."

All organisms met with in the conjunctival sacs disappeared after 48 hours of aureomycin therapy.

Conclusions

It will be seen from Tables 1 and 2 that we observed no improvement in any of the cases under treatment. Our results therefore are contradictory to those of most other workers on the subject. There are several factors which we believe are responsible for this apparent paradox:

1. *Improper diagnosis.* Many cases of ordinary subacute or follicular conjunctivitis are stamped "trachoma" and are subsequently "cured" by a sulfonamide or an antibiotic.

2. *Too much stress* is laid on the subjective improvement which the patient experiences following treatment. It must be remembered that relief from photophobia, blepharospasm, itching, and discharge may

all be obtained following the use of mild antiseptic drops such as methylene blue (0.025 percent) or argyrol (10 percent) solutions. These subdue the secondary infection and bring about an amelioration of the symptoms, but this should not be interpreted as an improvement or cure of the trachomatous process.

3. *Certain strains of virus* may be sensitive to aureomycin and others not. The Lebanese virus may be more resistant than the virus of Uganda or Italy. With our present knowledge, however, this possibility cannot be dealt with adequately until more extensive research has been carried out.

4. *Treatment inadequate.* Finally, it is possible that the dose of aureomycin that we employed was inadequate and the duration of treatment (12 days) too short to effect a cure. These two factors were strictly dependent on our supplies of aureomycin, which, as mentioned before, were very limited. It is possible that with larger doses given over a longer period of time cure of trachoma may be achieved.

It is noteworthy that all organisms met with in the conjunctival sacs were promptly controlled and eliminated.

Summary

The new antibiotic, aureomycin, has been tried on 20 cases of trachoma. There was no significant change in any of the cases after 12 days of therapy.

Secondary bacterial invaders were promptly eliminated after 48 hours of therapy.

It is urged that, in experimental trials on trachoma, the choice of cases and the standardization of the criteria of cure are of paramount importance.

We mention four factors which may be instrumental in leading various observers to arrive at different conclusions regarding the efficacy of drugs in the treatment of trachoma.

It is not our intention to prove that aureomycin is ineffective against trachoma. Our observations merely permit us to conclude that, when administered as we have described for selected Lebanese cases, aureomycin does not seem to exert any effect on the lesions of trachoma.

We are indebted to the Lederle Laboratories for providing us with the aureomycin capsules, ophthalmic drops, and ointment which were used in conducting these experiments.

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Ophthalmic Miniature

The disease which I witnessed in Tipperary, I believe to be a modified form of that denominated Egyptian Ophthalmia; which I have seen, upon a large scale, at Cairo, and in other parts of the Levant; and which committed such ravages in the British army at the time of, and subsequent to, its occupation of Egypt in 1803.

W. G. Wilde, *London Journal of Medicine*, 1851.

THE USE OF MIOTICS IN THE TREATMENT OF NONPARALYTIC CONVERGENT STRABISMUS

A PROGRESS REPORT

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In a preliminary report[1] it was noted that miotics could be used with benefit in the treatment of convergent strabismus. In such cases with hyperopia the use of miotics was found helpful to avoid, delay, or supplement the use of glasses.

In that report it was noted that 81.8 percent of cases of the isometropic type[2] of convergent strabismus were helped or cured with the aid of miotics. Miotics were particularly helpful in those cases which were initially periodic[3] in type, or which became so during the period of treatment before the miotics were used. Eighteen of these cases were in need of further treatment following surgery. Surgery had already been advised in some of these cases. The use of miotics made further surgery unnecessary in 17 of these cases.

While cases of anisometropia with convergent strabismus were helped by miotics in the more ametropic eye, the number of such cases so treated is still relatively few. Cases of divergent strabismus with hyperopia treated with miotics gave little or no response.

This second report will be limited to cases of nonparalytic strabismus of the convergent isometropic type.[2] It covers 88 new cases plus the 44 cases of the isometropic type previously reported.[1] In 71 cases of the new series, the strabismus was periodic in type before the use of miotics. In 17 of the 88 cases, the strabismus was constant in type before the use of miotics.

Miotics were of distinct value in the treatment of strabismus of the periodic type in 66 of 71 cases or 92.9 percent.

As in the first series of cases the use of the miotic was considered to have "helped" in the treatment when the strabismus was absent or less frequent while using miotics, sometimes with the use of glasses, usually without such use.

The use of miotics was of definite value in six of the 17 cases of the constant type, or in 35.3 percent.

In the first series[1] and the present series, there was a total of 109 cases of the periodic type and 23 cases of the constant type of convergent isometropic strabismus. Of the 109 cases of the periodic type, 97 were helped by the use of miotics, or 89.0 percent. Of the 23 cases of the constant type, 11 cases, or 47.8 percent, were helped by miotics.

DISCUSSION

I have been using miotics in cases of strabismus since 1939 (Javal[4]). In some cases, miotics have been used more or less consistently for seven years without detrimental effects. In some cases floropryl alone has been used for four years without untoward effects.

I feel that the use of miotics substitutes peripheral accommodative action at near as well as at distance for accommodation of central origin. Accommodation may be said to be more responsive to less stimulation. In this way the "overflow" stimuli to the convergence is materially lessened.

The term "accommodative" type is usually considered to describe those cases of convergent strabismus made straight with glasses—those cases usually showing a moderate or high degree of hyperopia. The considerable number of cases in which the error of refraction is negligibly small suggests that accommodation is a factor regardless of the error of refraction.

That the onset of the convergent form of strabismus is so completely limited to the

early years (when focusing at the near area is beginning to become important) is no mere coincidence. If the term "accommodative" type is to be retained, it should be widened in its scope to include most cases of convergent strabismus, as all such cases have an "accommodative" factor.

The use of miotics definitely gives greater "coverage" than do glasses. In their action on the accommodation, miotics seem to have a broader application than glasses.

Miotics were also used in amblyopic eyes for the stimulating effect on accommodation. This therapy was based on the suspected weakness of accommodation in cases of amblyopia. Such weakness is not easy to demonstrate, as comparison of the amplitude of accommodation in the two eyes of an individual cannot be considered accurate when the visual acuity of the two eyes is not equal. In most cases, if the amblyopia had been treated, the method of treatment in itself has also stimulated the accommodation so that these cases, as a rule, do not show a definite or consistent difference in accommodation at a later age when the visual acuity is equal.

In certain of these cases, however, despite the demonstration of equal visual acuity for distance with correction, the amplitude of accommodation in the two eyes is still demonstrably different. In some of these cases, the visual acuity of the two eyes may be equal at distance with correction and markedly unequal at the same distance without correction despite the fact that the error of refraction is practically the same in the two eyes.

CASE 1

A girl, aged eight years, had straight eyes after treatment for amblyopia and surgery. Her atropine refraction was: R.E., +6.0D. sph. ⊃ −2.0D. cyl. ax. 170°; L.E., +5.25D. sph. ⊃ −2.75D. cyl. ax. 180°. Postcycloplegic examination showed: R.E., +4.75D. sph. ⊃ −2.0D. cyl. ax. 170°; L.E., +3.75D. sph. ⊃ −2.75D. cyl. ax. 180°. Corrected vision in the right eye was 20/40−; in the left eye, 20/50+. Uncorrected vision was 20/50 in the right eye; 20/200, in the left. Tests for the near point of accommodation gave irregular results.

CASE 2

A boy, aged 14 years, had straight eyes after surgery and treatment for amblyopia. Homatropine refraction showed: R.E., +5.25D. sph. ⊃ +2.0D. cyl. ax. 82°; L.E., +4.75D. sph. ⊃ +2.0D. cyl. ax. 100°. Postcycloplegic examination showed: R.E., +4.5D. sph. ⊃ +2.0D. cyl. ax. 82° = 20/25+; L.E., +4.5D. sph. ⊃ +1.75D. cyl. ax. 98° = 20/25. Unaided vision was: R.E., 20/30; L.E., 20/200. With correction, the near point of accommodation was: R.E., five inches; L.E., six inches.

In my experience, cases of strabismus "cured" by one method or another showed relatively poor convergence amplitude (positive and negative convergence reserve) when tested shortly after the eyes became straight. These same cases, when similarly reëxamined one and one-half to two years later showed normal readings for their positive and negative reserve convergence at distance and at near. Such normal readings were rare or absent before an interval of one and one-half years from the time the eyes were straight, whether they were straightened by glasses, surgery, or other means. Such findings explain the tendency for recurrences after apparent cures.

CASE 3

A girl, aged 11 years, had straight eyes following surgery. Muscle tests at 13 inches (August 31, 1950) three months after the eyes were straight showed 20 prism diopters of exophoria. The negative reserve convergence was 26 prism diopters. Positive reserve convergence was 0 to minus-4 prism diopters. Sursumduction: right = left = 4.

Homatropine refraction at this time was: R.E., +1.25D. sph. = 20/20; L.E., +0.75D. sph. ⊃ +1.25D. cyl. ax. 90° = 20/20−.

One year later, the near muscle readings at 13 inches were: exophoria, 16 prism diopters; negative reserve convergence, 28 prism diopters; positive reserve convergence, 20 prism diopters. Sursumduction: right = left = 3 to 4$^{\Delta}$.

When miotics give good immediate results, they must usually be continued. The period of observation of cases of strabismus must extend approximately two years from the time the eyes are straight, and this period of observation must be extended if the patient has not yet reached the age of eight years. The fusion faculty must have a chance to mature,[8] thus preventing recurrences when the miotic is stopped.

In some cases, there was no recurrence of the strabismus when the miotic was stopped six months or one year after its use even though the patient was three or four years of age. In a considerable number of cases, however, there were recurrences within a short time after discontinuance of the miotic. These cases promptly responded to further use of the miotic.

Miotics are of little or no value if the miotic used does not have a deep and prolonged effect on the accommodation. For this reason, I have found that although the use of pilocarpine, eserine, or prostigmine[6] is helpful in some cases, more frequent applications of these drugs are required.

In my experience, floropryl[7] is by far the most efficient miotic for use in these cases. This product appears on the market in 0.1-percent strength in peanut oil. This strength or even one-half this strength tends to be irritating and at times painful. However, using this drug in 0.01-percent, 0.02-percent, or 0.03-percent strengths tends to eliminate such complications. Occasional redness is still found following its use but is easily controlled by cold compresses.

Only two cases of sensitivity to this drug have been noted in over 300 cases in which it was used for various reasons. In one case there was a definite local reaction with follicular conjunctivitis. In the second case, after nightly use of 0.01-percent solution of floropryl for four days, the child had nausea, wet the bed, staggered slightly and, on awakening the next day, yawned and stretched frequently. On discontinuing the drops, the symptoms disappeared.

In cases in which miotics were used before accurate vision could be determined, amblyopia was considered to be a negligible factor when the strabismus was either alternating or periodic in type. It was found that miotics were of little or no value insofar as their effect on the strabismus in cases with unequal visual acuity was concerned. The degree of equality in vision cannot be too strongly emphasized. Despite individual tolerances to unequal vision, the attempt must be made to obtain equal visual acuity for the best results.

In two cases reported earlier[1] as "helped," the eyes remained straight without glasses or drops. In one case reported earlier[1] as a failure with pilocarpine, the use of floropryl resulted in straight eyes. In nine of the 88 new cases, floropryl gave improved results in cases which were failures when weaker miotics were used. The need for less frequent use and less constant supervision when floropryl is used makes it a more practical miotic.

The following cases illustrate the use of floropryl in the treatment of convergent strabismus:

CASE 4

J. G., a girl, was first seen by me at the age of seven years. There was a history of the onset of a left periodic convergence at the age of two years. The patient had had orthoptic training for more than one year and treatment for amblyopia, using atropine in the right eye, for at least six months.

Vision without glasses was: R.E., 0.8; L.E., 0.8. The cover test showed a left convergence of 10 degrees. Refraction with atropine showed: R.E., +1.0D. sph. $\bigcirc$ +0.5D. cyl. ax. 165°; L.E., +1.25D. sph. The fundi and media were normal.

On April 28th, floropryl (0.03 percent) was prescribed for nightly use in each eye. On May 12th, the eyes were straight by cover test at distance and near. By June 16th, the patient was doing well. There was no convergence. On August 17th, the eyes were straight by cover test.

The patient was not seen for eight months. She returned with the history of using no miotic for four to five months and, within the last two or three weeks, a recurrence of the strabismus had been noted. The need for constant use of miotics for one to two years was explained and floropryl (0.03 percent, nightly) was again started. No trouble was reported one month later.

CASE 5

J. R., a girl, aged four years, had had no previous examination. There was a history of periodic convergence, more noticeable in the left than in the right eye, since the age of one and one-half years. The family history revealed many cases of strabismus.

Examination showed left periodic convergence. Atropine refraction: R.E., +1.5D. sph. $\bigcirc$ +1.0D. cyl.; L.E., +2.0D. sph. $\bigcirc$ +0.75D. cyl. On the illiterate chart, vision in both eyes was 0.6.

On July 29, 1949, the patient was started on 0.05-percent floropryl, one drop every other night.

By August 19th, it was reported that there was less frequency of "turning," and administration of the drops was increased to every night. There was still occasional convergence on August 29th, and glasses were prescribed on September 2nd. Floropryl was continued.

By October 20th, the eyes were straight by the cover test. On December 22nd, there was no change. February 23, 1950, the use of glasses was discontinued but the drops were continued. The patient was doing well without glasses when seen on April 8th. Examinations during the period from June 22 to December 19, 1950, showed that she was doing well on floropryl used only one time weekly. However, on April 14, 1951, a tendency toward recurrences was noted and floropryl was increased to nightly use. By June 4th, she was doing well.

CASE 6

T. S., a boy, aged three and one-half months, had a history of alternating periodic strabismus. His sister also had strabismus.

There was a right convergence of 15 to 20 degrees. Atropine (0.25 percent) was prescribed for use in the left eye daily for two weeks. Atropine was then stopped. Refraction when the child had reached the age of seven months revealed: R.E., +0.5D. cyl. ax. 90°; L.E., 0.5D. sph. Floropryl (0.01 percent) was ordered every other night. No change was noted in two weeks and the floropryl was discontinued. Since accommodation is not considered active at this early age (Mann[8]), it may be assumed that accommodation plays little or no part in the overactivity of convergence and that floropryl or other miotics will have little or no effect.

CASE 7

B. R., a girl, aged three and one-half years, had had left periodic convergence for two weeks. Examination showed this convergence to vary from 0 to 25 degrees. Atropine refraction revealed: R.E., +5.5D. sph.; L.E., +6.0D. sph. Glasses were not ordered but she was told to use 0.05-percent floropryl nightly in each eye. Nine months later, the mother reported that there was no squint so long as the drops were used. Examination at that time revealed no evidence of strabismus by cover test.

CASE 8

E. L., a boy, aged 14 months, had a history of left periodic convergence since the age of 10 months. There was a family history of strabismus but no history of illness or injury.

On examination there was left convergence of 0 to 45 degrees. Motility was normal. Atropine refraction showed: O.U., +5.0D. sph., approximately. The child cooperated poorly. One drop of two-percent pilocarpine in each eye twice daily was ordered. One month later there was questionable improvement. The drops were increased to every four hours. There was definite improvement but administration of the drops proved to be too much trouble.

At this time, 0.1-percent floropryl was prescribed, to be used every two days. No strabismus was noted for six months, at which time the mother stated that the left eye "turned in" occasionally. Atropine sulfate (0.5 percent) was ordered for the right eye; floropryl was continued in the left eye. At the end of two weeks, the atropine was discontinued and floropryl was ordered for each eye.

The eyes remained straight for one year with floropryl (0.05 percent) every two nights. When seen at that time, the mother said that the left eye turned in occasionally. Floropryl (0.03 percent) was ordered twice daily in each eye. The eyes were straight when the child was seen five months later. Drops were still being used.

This is an example of a child, aged 14 months, being treated without glasses for over two years.

CASE 9

B. H., a girl, aged six years, had had right periodic convergence since the age of five years. For six months before she was first seen, she had been receiving the Bates treatment. The mother thought her condition was worse. No glasses had ever been worn.

Examination showed the right eye to be convergent more of the time than straight. Vision was: R.E., 0.4; L.E., 0.8, without glasses. Atropine refraction revealed: R.E., +6.75D. sph. $\bigcirc$ +0.75D. cyl. ax. 110°; L.E., +6.75D. sph. $\bigcirc$ +0.5D. cyl. ax. 40°. Under atropine the eyes were parallel. Glasses with one diopter less than the atropine findings were ordered. With glasses, the eyes were straight; without glasses, there was a right convergence. Vision with glasses was: R.E., 0.5; L.E., 0.6.

Floropryl (0.03 percent) was ordered nightly. The eyes were straight with and without glasses on examination six weeks later. Within three months, vision with glasses was: O.U., 1.0. The patient was told to use glasses for school work and floropryl (0.01 percent) nightly. Further follow-up for seven months showed no convergence.

CASE 10

E. B., a girl, was first seen by me at the age of five years. Her history showed an onset of left periodic convergence at the age of four years. There was no history of illness or accident. The family history was negative. No glasses had been worn.

With the illiterate E, vision was: R.E., 0.6; L.E., 0.5. Atropine refraction showed: R.E., +6.25D. sph.; L.E., +6.75D. sph. $\bigcirc$ +0.25D. cyl. ax. 90°. The fundi and media were normal. No glasses were ordered. Nightly use of floropryl (0.02 percent) controlled the strabismus over an observation period of six months.

CONCLUSIONS

Since we do not know (Brown and Kronfeld[9]) the true effect of the wearing of glasses on the growth of the eyeball, it is

wise to have an open mind toward any method which offers the possibility of delaying or avoiding the use of glasses in cases of hyperopia. The use of miotics may give this opportunity.

Miotics may be used as a substitute for glasses in very young patients, in those with behavior problems, and in cases in which other psychologic factors argue against the prescribing of glasses.

When glasses have been worn and are suddenly removed, the effect on accommodative effort is marked, and miotics may be used to help make the change from glasses to no glasses a gradual one.

The use of miotics may be helpful in the treatment of amblyopia where an unsuspected lack of accommodation may be present.

The consistency and speed with which miotics without the use of orthoptic training help the strabismus and replace glasses in the periodic type suggest that the use of a miotic may effectively replace orthoptics in many such cases. With miotics the beneficial effect is maintained throughout the waking hours, which is not true of orthoptics. In cases helped by miotics, orthoptics may possibly help to prevent later recurrences. This phase should be explored.

Miotics, if properly used as suggested herein, should prove a definite aid in the treatment of convergent strabismus, especially in the periodic type with or without a considerable amount of hyperopia.

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Ophthalmic Miniature

Socrates proceeded: I thought that as I had failed in the contemplation of true existence, I ought to be careful that I did not lose the eye of my soul; as people may injure their bodily eye by observing and gazing on the sun during an eclipse, unless they take the precaution of only looking at the image reflected in the water, or in some similar medium (Phaedo-Plato).

EXPERIMENTAL STUDIES WITH A MEMBER OF THE PARACOLON ESCHERICHIA GROUP PRODUCING HUMAN PANOPHTHALMITIS

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The history of gram-negative bacilli associated with human eye infections is well known. Members of the Hemophilus,[1, 2] Pseudomonas,[3] Escherichia,[2] Brucella,[4] Klebsiella,[5] Shigella,[2, 6] and Pasteurella[7] genera have been listed and described as causative agents in diverse pathologic conditions of the eye in humans. We have been unable to find any reports in the literature which attribute a human eye infection to paracolon bacteria, although two instances of eye infections caused by paracolon organisms in turkeys and canaries have been observed.[8]

The exact status of paracolon bacteria has not as yet been clearly established. Although they have been granted a genus designation,[9] many of their biochemical and antigenic characteristics are closely related to those of the coliform or Salmonella bacteria. However, bacteria belonging to recognized groups within the Paracolon genus can be identified by their serologic specificity[10, 11] and by their usually slow fermentation of lactose, with or without the formation of gas.

Paracolon bacteria have been reported as causative agents for gastroenteritis[10, 11] and pneumonia[12] in humans, as well as for the eye infections in turkeys and canaries already mentioned.

CASE REPORT

History. On July 22, 1950, a 65-year-old white woman was seen in consultation because of chemosis and edema of the left eye. She had previously been admitted to another hospital because of a 10-day period of excess vomiting and diarrhea, which had been so severe that her family physician was apprehensive about her electrolyte balance.

When first seen in the eye department, she was in an acute state of mental confusion, complaining of pain in the epigastrium and a smothering feeling about her heart. The left eye was so chemotic that tension could not be taken and the eye felt stony hard to finger palpation.

She had been well until 10 days prior to her admission and at this time it was believed that the abdominal complaints were secondary ones resulting from an acute glaucoma. The patient was then transferred to the eye and ear hospital with a tentative diagnosis of acute glaucoma and treated with drops containing one-percent eserine, two-percent pilocarpine, and three-percent dionine. The following day her condition grew worse.

She at no time complained of pain in the eye, although by this time the chemosis was so severe that she was unable to close her lids, and the chemotic conjunctiva protruded. At this time, a diagnosis of panophthalmitis was made. She had a numbness of her cheek and tongue with impairment of speech, her mental confusion becoming more marked.

The history of her past health, as obtained from her daughter, showed that she had had six children and a normal menopause. She had had a cholecystectomy some years ago, in addition to some type of operation for a "brain abscess" following which she had had frequent headaches. Ten months prior to her present illness she had had an episode of vomiting and pain in the abdomen somewhat similar to the present complaint.

Physical examination. The patient was a well-developed, obese, white woman, aged 65 years, who appeared acutely ill. Blood pressure was 180/90 mm. Hg. The abdomen showed an active peristalsis, but the re-

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mainder of her physical examination (excepting that of the left eye) was negative. Temperature was 98.6°F., pulse 70, and respiration, 22. Urinalysis and blood chemistry were within normal limits. The only abnormal laboratory finding was that white blood count was 15,700 with 73-percent polys. The left eye was edematous, chemotic, and stony hard.

Clinical course. On the second day of her admission her temperature rose to 100°F., the only time it was ever above 98.6°F. Since the patient was so acutely ill, it did not seem likely that her condition was due solely to the ocular complaint. X-ray studies made of her chest, sinuses, and skull showed no abnormalities, and a flat plate of her abdomen yielded similar results. It was felt that she was too ill to withstand barium studies of the gastro-intestinal tract. She was, therefore, treated with antibiotics consisting of penicillin, aureomycin, and chloromycetin, and was fed by intravenous fluids.

On August 3, 1951, it was felt that her condition had improved enough so that she could undergo an enucleation of the left eye. On that date, under pentothal intravenous anesthesia, an enucleation was started. Immediately following the circumcorneal incision, much pus presented in the operative field; this apparently came from the upper portion of the orbit under the conjunctiva. The operative procedure was then changed to an evisceration. As soon as the cornea was incised at the limbus the same thick, yellow, creamy pus was seen to fill the globe. It is of interest to note that there was no particular odor to the pus.

After the evisceration had been completed it was seen that the globe had ruptured superiorly under the superior rectus muscle, but Tenon's capsule apparently limited its extension. A drain was inserted into the eye as well as into the orbit. The patient made a surprising recovery and left the hospital on the fifth postoperative day. She is now well and has no complaints.

Experimental procedures and results

Biochemical studies

A culture from the thick, creamy pus which filled the globe was made into infusion broth and streaked on a blood agar plate.* Individual colonies on the plate were raised, opaque, had an even, slightly granular surface, and were somewhat irregular at the periphery. The broth culture yielded similar colonies when streaked on infusion agar plates. The individual organisms were approximately 1.0 to 1.5 μ by 0.3 to 0.4 μ, stained gram negative, and were not motile. Biochemical and serologic studies on sample colonies indicated that the pus had yielded a pure culture.

Fermentation studies were made in nutrient broth (Difco) tubes containing one-percent concentrations of Seitz-filtered carbohydrates plus indicator. The broth was adjusted prior to autoclaving so that the final pH after addition of the carbohydrate was 7.1. Both parafilm-sealed and unsealed tubes containing inserts were used in all the biochemical tests. The tubes, containing a total volume of 5.0 ml., were observed daily for 30 days at 37°C. Twenty-four hour cultures, grown on nutrient agar slants, were washed off with sterile physiologic saline, resuspended in saline, and adjusted to a concentration of approximately 1.0 by 10^{-8} cells per ml. Each broth tube received a 0.1-ml. inoculum of this suspension of cells.

The results indicated that arabinose, dulcitol, galactose, glucose, levulose, maltose, mannitol, rhamnose, sorbitol, and xylose were readily fermented to acid and gas within 24 to 36 hours in both the sealed and unsealed tubes. Acid was produced in the lactose tubes in approximately 65 hours, while the first bubble of gas became apparent only after five days' incubation. After 30

* The initial identification which placed the organism in the genus Paracolon was performed in the laboratory of the Presbyterian Hospital of Pittsburgh.

days' incubation, approximately 25-percent gas was formed in both the sealed and unsealed lactose tubes. Dextrin, inositol, raffinose, salicin, and sucrose gave alkaline reactions. Litmus milk was acidified. Gelatin was not liquefied. The organisms were indole positive, methyl-red positive, Vogues-Proskauer negative, and Koser's citrate negative. Tests for urease and H_2S were negative.

A comparison of these results with the classification of Schaub[13] would place this organism* with the Paracolon escherichia.

Serologic studies

The patient's serum was subjected to a series of agglutination tests with the organism isolated from her left eye, and also with a variety of antigens† representing members of the gram-negative group. All antigens with the exceptions of Salmonella typhosa "O" (phenol 0.5 percent) were formalin (0.5 percent) killed. The tests were conducted at 56°C. and recorded after 20 hours' incubation. Table 1 summarizes the results obtained. The specificity of the isolated organism for the patient's serum is apparent. Normal serum and saline control studies were negative.

Toxicity studies

A 24-hour agar slant culture was suspended in sterile physiologic saline. Varying dilutions of the suspension were prepared, and plates were poured for total viable counts. White mice weighing 30 gm. (± 2.0 gm.) each were inoculated intraperitoneally with a 1.0-ml. suspension of the organisms. In all instances in which death occurred, the mice were autopsied and cultures mades from the heart blood.

Brain-heart infusion broth, eosin-methylene blue agar plates, and S-S agar plates were used for culturing. Typical colonies on the plates were selected for biochemical studies. All autopsied animals yielded organisms identical with those originally isolated from the patient's eye.

The remaining mice were observed for 10 days and then killed. No bacteriologic examination was undertaken on the latter group. Our results are summarized in Table 2. Of the nine mice that died, only one survived as long as 23 and one-half hours. All the others died within 12 hours.

TABLE 2

MOUSE-TOXICITY REACTIONS WITH A CULTURE OF P. ESCHERICHIA ISOLATED FROM A HUMAN EYE

No. of Organisms per ml.	Inoculum (ml.)	No. of Mice Inoculated Intraperitoneally	No. of Deaths
10×10^8	1	3	3
40×10^6	1	6	6
16×10^6	1	6	0
16×10^4	1	6	0
16×10^2	1	6	0

TABLE 1

AGGLUTINATION TESTS WITH PATIENT'S SERUM

Antigen	P. Escherichia from Eye	S. Typhosa "O"	S. Typhosa "H"	Flexner V	Flexner Z	Shigella Sonnei	Controls† Negative
Titer	1:5120*	1:40	1:20	1:80	1:80	1:80	

* Figures indicate highest dilution at which agglutination occurred.
† Normal serum and physiologic saline.
Note: There was no agglutination in 1:10 dilution of patient's serum with S. para A, S. para B, Pasteurella tularensis, Brucella abortus, Proteus OX19, and Flexner W. The antigen designations are those employed by the Pennsylvania Department of Health.

* We wish to thank Dr. M. Frobisher, Jr., and Dr. W. H. Ewing for the serologic confirmation of this organism. Their report from the Enteric Bacteriology Laboratories at Chamblee, Georgia, classified the organism as "Paracolon escherichia, belonging to E. coli O-group 62."

† The antigens were prepared at the Bureau of Laboratories, Pennsylvania Department of Health, Philadelphia 4, Pennsylvania.

Rabbit-eye studies*

A group of male albino rabbits weighing approximately 3,500 gm. each were inoculated with a 24-hour suspension of the organism isolated from the patient's eye.

Two methods were used for this purpose. In one series, the cornea was scratched linarly with a 25-guage hypodermic needle. Three drops of the suspension were then instilled into the conjunctival sac. In the second series, after the animal had been anesthetized with intravenous nembutol, the conjunctiva was grasped at the limbus on the nasal side. A Wheeler knife needle was then used to make an oblique track in the cornea at the temporal side. The knife was withdrawn, a 26-gauge hypodermic needle on a tuberculin syringe was inserted into the anterior chamber via the track, and 0.02 ml. of suspension was injected into the anterior chamber.

Six rabbits were inoculated, three by each of the two methods.

The eyes of the rabbits inoculated by corneal scratch remained normal even after a week. In this group, cultures taken both from the conjunctival sac and the anterior chamber seven days after inoculation did not show presence of the paracolon organism.

The eyes of the rabbits inoculated in the anterior chamber developed a conjunctivitis together with an opacity of the cornea within 48 hours. Seven days after inoculation, cultures taken from the conjunctival sac and the anterior chamber of the eyes of two rabbits showed presence of the paracolon organism. Cultures taken from the heart blood at the same time were negative. The third rabbit was surveyed after 14 days, and no evidence of the organism was demonstrable.

In both groups of rabbits, eyes not inoculated remained normal.

A rabbit receiving inoculations of a culture of Escherichia coli comparable in numbers to the paracolon suspension, both by corneal scratch on the right eye and in the anterior chamber of the left eye, remained normal for seven days.

All animals were killed after stated periods of observation.

Antibiotic studies

Penicillin (buffered crystalline penicillin-G potassium—E. R. Squibb and Sons), aureomycin (parenteral aureomycin HCl—Lederle Laboratory), streptomycin (base as streptomycin sulfate—Winthrop-Stearns), chloromycetin (chloramphenicol, diagnostic, for investigational use—Parke-Davis and Co.), and terramycin (crystalline terramycin hydrochloride—Chas. Pfizer and Co., Inc.) were employed in the assay-disc method for evaluating the sensitivity of the P. escherichia isolate to these antibiotics.

Twenty-four-hour agar slant cultures were suspended in sterile saline, and 1.0 ml. of the suspension was inoculated into an Erlenmeyer flask containing 200 ml. of brain-heart infusion agar. The resulting count of organisms was approximately 2.0 by 10^{-7} cells per ml.

After plates were poured and allowed to harden, analytical filter paper discs (12.7 mm. diameter—Schleicher and Schnell, New York) were placed on the surface, and 0.08 ml. of freshly prepared antibiotic dilutions were then pipetted onto the discs. Plates were incubated at 37°C. and recorded after 24 hours' incubation. Only complete inhibition zones through the agar were measured.

Results recorded in Table 3 indicate that the order of diminishing activity against the P. escherichia is terramycin, chloromycetin, aureomycin, streptomycin, and penicillin.

Discussion

The results presented in this survey indicate that the organism isolated from a human eye played a causative role in the pathologic condition of the eye. The relatively high titer (1:5,120) of the patient's serum to this or-

* We acknowledge our indebtedness to the Addison H. Gibson Laboratory for the use of their facilities in the eye studies.

TABLE 3

ANTIBIOTIC REACTIONS AGAINST P. ESCHERICHIA ISOLATED FROM A HUMAN EYE

Penicillin		Aureomycin		Chloromycetin		Terramycin		Streptomycin	
Units/ml.	Zone of Inhibition	mg./ml.	Zone of Inhibition	mg./ml.	Zone of Inhibition	mg./ml.	Zone of Inhibition	mg./ml.	Zone of Inhibition
1×10^5	2.0 mm.	0.5	4.5 mm.	0.1	3.0 mm.	0.05	4.5 mm.	5.0	4.0 mm.
5×10^5	±*	0.25	3.0 mm.	0.05	1.5 mm.	0.025	3.5 mm.	2.5	3.0 mm.
25×10^5	−†	0.125	2.0 mm.	0.025	±	0.012	2.5 mm.	1.25	2.0 mm.
		0.062	1.0 mm.	0.012	−	0.006	1.0 mm.	0.625	1.5 mm.
		0.031	±			0.003	±	0.312	1.0 mm.
		0.015	−					0.156	±

* Partial inhibition at surface of agar, but no clear area through the agar.
† No inhibition.

ganism is especially significant as evidence substantiating this observation. The patient's serum showed a titer of 1:160 against P. escherichia 15 months after release from the hospital, which fact would serve as additional evidence for the etiology of the infection. Since most human illnesses attributable to paracolon bacteria have been associated with gastro-enteric infections, it seems possible that this patient's enteric malady and her subsequent eye infection may have been caused by the same paracolon organism.

Maxwell[6] reported six cases of iritis occurring in patients suffering from enteric illness attributable to Shigella dysenteriæ, although he did not report isolating this organism from the affected eyes. Obviously, additional research should be undertaken on the possible association between the enteric infections and the pathologic conditions of the eye before any conclusions can be drawn concerning the potential pathogenic roles of P. escherichia.

Although no marked antigenic relationship exists between the P. escherichia and members of the Shigella and Salmonella genera as reported here, it is interesting to note that minor relationships between members of the paracolon group and certain Salmonella[14] and Shigella[15] have been reported in the literature.

The studies conducted on rabbit eyes were intended only as a preliminary survey. A more systematic approach to this problem is contemplated.

SUMMARY

1. A pure culture of Paracolon escherichia was isolated from a human case of panophalmitis. The causative role of this organism was further substantiated by serologic evidence of a high titer of the patient's serum to P. escherichia.

2. This organism was toxic for white mice on intraperitoneal inoculation of approximately 40 by 10^6 cells.

3. The paracolon organisms gave rise to a conjunctivitis and opacity of the cornea when inoculated into the anterior chamber of rabbits' eyes.

4. Results with the assay-disc method indicate that the order of diminishing activity against the P. escherichia is terramycin, chloromycetin, aureomycin, and penicillin.

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SURFACE THERAPY WITH THE SHAHAN THERMAPHORE IN RETINAL DETACHMENT*

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Since 1929, the principles of Leber and Gonin have been recognized as the basis for any retinal detachment surgery. Leber discussed the importance of the retinal tear in permitting the undermining of the retina by the liquid part of the vitreous; Gonin pointed out the possibility of returning the retina to its place and holding it there with adhesions produced by the thermocautery.

The numerous surgical methods devised since 1929 have all been directed toward two main objectives—evacuation of the subretinal fluid, to return the retina to normal position and production of sufficient exudate to produce adhesions which will hold the retina and close the tear.

In 1934, Wheeler, and also Crisp, suggested that the Shahan thermaphore in conjunction with posterior sclerotomy might meet these requirements. In 1935, Langdon reported that the first case treated by this method was a complete success; in 1938, he reported a total of five cases, four of which were successful.

In 1936, Shahan used his thermaphore for retinal detachment but made no report because the patient died a week after operation. However, he was certain that the retina was attached at the time of death. In 1939, Post reported 10 cases of which seven were successful.

We have used the Shahan thermaphore in retinal detachment surgery at the Pennsylvania Hospital since 1940, modifying our technique as experience dictated. This report is based on 14 cases of simple retinal detachment operated upon by Dr. Alexander G. Fewell, by Dr. M. Luther Kauffman, or by me. Of these cases, nine were successful. Our criteria of success were: (1) Complete reattachment, (2) full or nearly full field, and (3) the return to normal vision or to the vision the patient had before detachment.

As soon as a diagnosis of serous detachment was made, the eye was atropinized, pinhole goggles were prescribed, and the patient was admitted to the hospital for complete bedrest for several days prior to operation. At first we used local anesthesia but, recently, intravenous sodium pentothal has been used in all cases.

A conjunctival incision exposes the area of the globe over the detachment after a final ophthalmoscopic examination has fixed in mind the limits of the detachment and the

* Presented before the College of Physicians of Philadelphia, Section on Ophthalmology, October, 1951.

position of the retinal tear. It has been suggested that this conjunctival incision is unnecessary and that may be true. However, applying the thermaphore directly to the conjunctiva would produce adhesions which might complicate any future surgery.

An applicator, whose terminal end is a grid one-cm. square, is inserted in the thermaphore and maintained at a constant temperature of 160°F. After all bleeding has been checked and the operative area made as dry as possible, the grid is held firmly against the sclera, the region of the ciliary body being carefully avoided, for periods of one minute until such applications have covered the entire scleral area corresponding to the detachment. This may require three to 10 applications, depending on the extent of the detachment. In the neighborhood of the retinal tear, the applications are overlapped in order to obtain a greater reaction at this critical spot.

The construction of the applicator makes it easy to reach the posterior portions of the globe and to cover areas beneath the extraocular muscles without cutting the muscles from their insertions. Post resected muscles in some of his cases but Langdon never did. Until recently we cut muscles in some of our cases but now we simply elevate the muscles with a strabismus hook and pass the thermaphore underneath.

Great care must be taken that neither the grid nor the shaft of the applicator remains in contact with the lids during the minute of application or first- or second-degree burns will result.

The removal of subretinal fluid is achieved by piercing the sclera over the position of the retinal tear with a von Graefe cataract knife the blade of which, about 2.5 mm. in width, is turned at right angle to the incision. After the escape of the fluid, the knife is turned back to its original position so that in withdrawing the blade no other incision is made.

In most of our cases one such puncture was made after the heat had been applied. However, when the detachment was extensive and elevation great, two punctures were made—one before and one after the heating process. One puncture was always made at the point of the tear and the other where we felt drainage was most needed.

In a case which required two operations before success, one puncture was made at the conclusion of the first, and unsuccessful, operation, and one puncture was made at the beginning of the second, successful operation. The course in this case, which was one of extensive detachment, suggested that puncture before heat might be better in some cases. When I next encountered a similar problem, I used a puncture both before and after the heating and got an excellent result.

Post used two punctures three-mm. in length before applying the thermaphore; Langdon, two or three punctures before applying the thermaphore; Shahan made three trephine holes in the sclera before heat and performed a posterior sclerotomy afterward.

The disadvantage of sclerotomy at the earlier stage is that one must then work with a soft globe; the advantage is that with a soft globe it may be easier to reach the posterior of the globe, if this is necessary.

Naturally the fewer punctures made the less likely that vitreous hemorrhage will occur as a complication. The conjunctiva is closed with one or two running sutures.

We did not dress the eyes in our cases for two days at which time a moderate degree of chemosis was usually found. Complete bedrest and binocular bandaging were practiced for two weeks, after which pinhole goggles were used; the head of the bed was gradually elevated through the third week. The patients were released from the hospital in the fourth week but kept on pinhole goggles for another month and on restricted activity for six months.

After his first use of the thermaphore, Langdon, in 1935, expressed the opinion that this operation was only applicable to low detachments of small extent. Our experience, however, suggests otherwise. Four of our nine successful cases had detachments involving at least one half of the retina, and three of these exhibited an elevation at the highest point of 10 to 15 diopters. Only one

of these three required two operations.

In this small series the finding of a retinal tear seemed to have no bearing on the results obtained. In our nine successful cases, we found a tear in five and a disinsertion in one; tears were found in three of our five failures.

No relapses have occurred in our nine successful cases, the most recent of which was operated upon in December, 1950. These nine patients all had complete reattachments. Seven patients can now read the 20/20 line or part of it; one patient who, because of myopic degeneration, had 20/70 in each eye before detachment has returned to 20/70; the other whose vision was 20/50 in each eye before detachment, because of congenital amblyopia, has returned to 20/50.

The peripheral fields were full in three cases; in five, the constriction was between 10 and 20 degrees in the area corresponding to the thermaphore application; in one case there were 30 degrees of such constriction. This was surprising because of the presence of heavy retinal pigmentation in the area affected by the thermaphore.

In two of our five failures, we believe there were extenuating circumstances. One case was that of a woman who came to the hospital two months after receiving a blow in the eye. There was extensive detachment, with two large tears and a dislocated lens. The lens was extracted with a loop and a thermaphore operation done; one month later the thermaphore was applied a second time. However, the patient, an alcoholic of low intelligence, refused to lie in bed. She became totally blind.

In another instance, a man developed neurotic anxiety after operation, tossed about in bed, refused a second operation, and was referred to a psychiatrist. When a patient is restlessly searching his mind in an effort to determine what action of his past life has brought this judgment upon him, he makes a poor prospect for any retinal detachment surgery.

In our hands, then, this method has given 75-percent success in coöperative subjects.

The advantages over other methods seem to be:

1. There is less trauma to the eye.

2. Since a large area is affected by a single application, the retinal tear need not be so accurately localized.

3. For the same reason large holes and multiple holes are treated more effectively.

4. If the tear has not been found, or there is a second undiscovered tear, an excellent chance of closure still remains, since the thermaphore is applied over the entire area of detachment.

5. There is a uniform reaction throughout with no variability in temperature or depth of penetration.

6. When the muscles are not cut, there is no danger of subsequent muscle imbalance, an infrequent but very annoying complication.

7. The thermaphore may be used prophylactically to reduce the danger of detachment after any posterior sclerotomy, such as one made for the removal of an intraocular foreign body by the posterior route.

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NOTES, CASES, INSTRUMENTS

DEMONSTRATION EYEPIECE FOR THE SLITLAMP

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The demonstration of some detail at the slitlamp to another observer is often rather difficult because, due to the high magnification, the slightest movement of the patient's eye gets the object of interest out of focus. This is particularly true in teaching comparatively inexperienced observers.

At my suggestion, the Bauch & Lomb Optical Company constructed the instrument presented here which enables two observers to see the same object simultaneously. A device similar to that used for the big Gullstrand ophthalmoscope was used as the base. It consists essentially of a tube that can be inserted into one side of the binocular microscope of the slitlamp, an arrangement of prisms which doubles the image, and two tubes emerging at an agle each containing an ocular (fig. 1).

Fig. 1 (Stocker). Demonstration eyepiece for slitlamp with pointer, before being inserted into the microscope.

After inserting the observation eyepiece into the microscope, the observer focuses the microscope on the site of interest, using the pointer as a guide for the second observer. The latter, using the second tube for observation, will then be able to see exactly the same object the first observer is pointing at. Of course, only monocular vision is possible.

Fig. 2 (Stocker). Demonstration eyepiece in place. The first observer is directing the pointer with his left hand toward the object to be studied in the field of the ocular. The second observer is looking through the tube of the demonstration eyepiece which emerges at an angle from the main tube.

1110 West Main Street.

THE TWO PHASES OF ABNORMAL RETINAL CORRESPONDENCE

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The concept of and the term abnormal (anomalous) retinal correspondence* have led to a good deal of confusion among ophthalmologists. One reason for this confusion is that authors of books and articles on the subject have not clearly distinguished between the two phases of normal and abnormal retinal correspondence. Notable excep-

* At a recent ophthalmological meeting at the New York Academy of Medicine the speaker during the instruction hour said that some 90 percent of strabismus patients have abnormal retinal correspondence. In the ensuing discussion one ophthalmologist said that there is no such thing as abnormal retinal correspondence. Evidently they were not referring to the same thing and some clarification is desirable.

tions are Swan, Burian, and one or two others. Let us now clarify this distinction in the simplest possible way.

The two phases of normal and abnormal retinal correspondence are:

1. Normally the two macular images of the same object are projected (that is, mentally referred) to the same point in space. If the macular images are not of the same object but are "compatible" images—such as could be seen in the same place, for example, bird and cage—the two images are superposed, That is, normally, we have what may be called bimacular superposition of compatible images.

In abnormal retinal correspondence the two macular images are not superposed, but separated. They are projected to different points in space. We thus have bimacular separation instead of bimacular superposition.

2. Normally, the macular image of the object of attention in one eye and the nonmacular image of the same object in the other eye placed there, say, by a prism, are projected to different points in space. Let us call the nonmacular image the paramacular image. Then normally the macular and paramacular images of the same object are projected apart from one another, that is, we have macular-paramacular separation. In abnormal retinal correspondence we have (more or less) superposition of the macular image in the fixing eye and the paramacular image in the deviating eye. There is even some evidence of a sort of fusion. At any rate we have macular-paramacular superposition.

The nonmacular region in the deviating eye which develops a sort of correspondence to the macular image in the fixing eye may be termed a comacula. This is more expressive than the term paramacula as it calls attention to the correspondence.

Thus abnormal retinal correspondence consists of (1) bimacular separation of images, and (2) macular-comacular superposition of images.

The former is diagnosed by the Bielschowsky after-image test, the latter by comparing the objective angle of squint with the subjective angle of squint. The difference between the two angles is the angle of anomaly and shows the extent of the abnormal correspondence. In an examination which involves testing for the two phases of possible correspondence or noncorrespondence we may have three possibilities:

1. Bimacular separation of images (abnormal correspondence) and macular-comacular superposition of images (abnormal correspondence). This would show that the abnormal correspondence is deep seated and probably impossible to remove by exercises.

2. Bimacular superposition of images (normal correspondence) and macular-comacular superposition of images (abnormal correspondence).

3. Bimacular separation of images (abnormal correspondence) and macular-comacular separation of images (normal correspondence).

The last two findings show that the abnormal correspondence is not so deep seated, and is more likely to be amenable to orthoptic exercises. In these latter, monocular diplopia as a step in the treatment is more easily aroused.

It is interesting to note that, in defining the terms, where this distinction is not clearly brought out some writers stress the phase of bimacular separation of images (Swan) and others stress the macular-comacular superposition of the images (British orthoptists, Keith Lyle). In outlining treatment also it is amazing how often the writers by not distinguishing between these two phases of normal and abnormal correspondence appreciably lessen the effectiveness of their suggested procedures.

The two terms which I have chosen to represent the two phases of abnormal correspondence—(1) bimacular separation of the images and (2) macular-comacular superposition of the images—seem to be appropriate and may well be generally adopted. I use

the term "macular" in the sense in which Walls* used it when he pointed out that textbook descriptions of the retina and of some of its landmarks may be incorrect. The true macula is only a little region of 2.0 to 2.5 degrees in the center of the fovea, the latter measuring some 6.0 to 7.0 degrees.

Whether the terms herein suggested are acceptable or not, the need for clarifying and specifically naming the two phases of abnormal retinal correspondence has certainly been too long neglected.

37 West 97th Street.

EYELASH IN THE UPPER LACRIMAL PUNCTUM

JOHN J. STERN, M.D.
Utica, New York

A man, aged 35 years, complained that his right eye was irritated and bloodshot. A few days previously a cloud of dust had blown into his eyes and he had rubbed the left eye vigorously. The marked discomfort which had followed did not subside.

The conjunctiva of the nasal quadrant was injected and an ulceration about one mm. in diameter was demonstrated on the plica semilunaris. The object causing this ulceration was found to be an eyelash which had entered the upper lacrimal punctum, root first, with the tip broken off. About one-half mm. of the eyelash stuck out, just enough to touch the plica semilunaris. The whole eyelash was nearly four mm. long.

It was removed, a bland ointment was applied, and the ulcer healed within two days.

Anatomically the cilium had no business to lodge itself in this peculiar position. The average length of the vertical portion of the canaliculus is 1.8 to 2.25 mm., but almost four mm. of the cilium had entered it. There is a physiologic constriction just above the punctum where the canaliculus is 0.1 mm. wide—much less than a average cilium.

What may have happened is that the eyelash fell out when the patient rubbed his eye, that its root was lying in front of the punctum, and that the whole eyelash was actively sucked into the canaliculus, thus demonstrating nicely a negative pressure in the tear sac as postulated by several theories of the conduction of tears.

3 Hopper Street.

CLEARING OF CORNEAL INFILTRATES WITH TOPICAL CORTISONE

GEORGE J. WYMAN, M.D.
Peoria, Illinois

A number of investigators have reported favorable response from topical cortisone in the treatment of epidemic keratoconjunctivitis. Since it became available, I have been using the drug for the treatment of this condition and all cases that I have seen early have responded well without formation of any corneal infiltrates.

Recently, I saw two late cases of the disease and the corneal involvement was well developed. Although it was felt that treatment would be of no benefit, since both patients had white eyes with no inflammatory symptoms, the visual results were so spectacular that it was felt that the cases deserved comment.

CASE REPORTS

The first case was that of a 23-year-old woman who had been treated for an eye infection for three weeks. She was first seen on August 25, 1951, at which time the vision in the right eye was 20/200, no Jaeger; the left eye, 20/30−3, with J1. There were no infiltrates in the left cornea but they were so numerous and dense in the central region of the right cornea as to appear almost as a disciform opacity.

Treatment with cortisone (5.0 mg. per cc., every hour) was instituted and on September 8, 1951, the vision in the right eye

* Walls, G. L.: Am. J. Ophth., **32**:1409-1410 (Oct.) 1949.

was 20/40−2. The opacities at that time had cleared remarkably. On September 29, 1951, she was seen again at which time the vision was 20/30 and only about four discrete small central opacities could be seen.

The second case was that of a 29-year-old woman who had been treated for three weeks with some antiseptic ointment. When seen on September 12, 1951, the vision was 20/50—2, J3, in the right eye; left eye, 20/20, J1. The right cornea showed many rounded subepithelial infiltrates. The left eye was clear. After two weeks of cortisone therapy, the vision in the right eye was 20/20−2, J1.

Comment

It is well known that eyes recently recovered from the acute manifestations of epidemic keratoconjunctivitis make good visual recoveries over a period of months. It has never been my experience, however, to see such prompt visual recovery within two weeks and I feel that the drug may have been responsible. The mode of action, of course, remains obscure since this seems to be the only viral disease in which it seems to have given beneficial results. It would be interesting to see if artificially induced opacities of the cornea would clear under cortisone therapy.

744 Jefferson Building (2).

AN INSTRUMENT FOR REMOVAL OF EYE SUTURES*

William J. Harrison, M.D.
Philadelphia, Pennsylvania

For a number of years I have employed cataract sutures. At times it has been most difficult to remove these sutures using the ordinary scissors and a pair of fixation forceps. At such times, a sudden movement of the eye has resulted in bleeding which necessitates a postponement of the effort, or may result in the forcible removal of the suture, again resulting in bleeding and sometimes a reopening of the wound. To overcome these embarrassing conditions, the present combined instrument is offered.

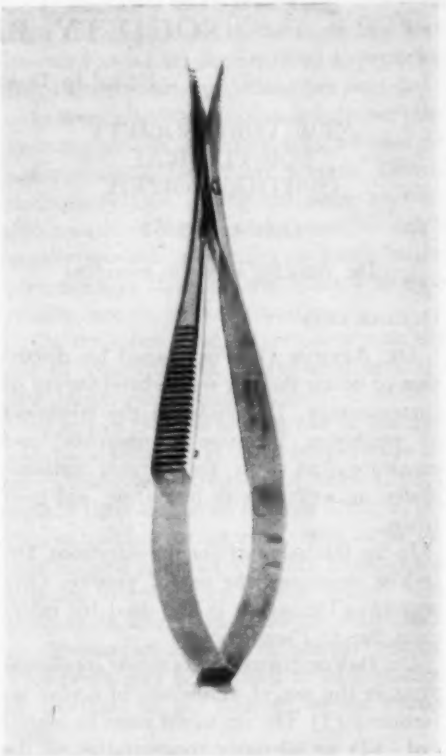

Fig. 1 (Harrison). Instrument for removal of eye sutures.

It consists of a pair of scissors with the usual blades, but sharply notched on its noncutting border. These notches start at three mm. from the point and extend a distance of three mm. along the border.

One blade of the scissors is introduced through the loop and the suture is cut. This blade is then rotated so that the knot is entangled in the serrations and the suture readily lifted out. This is a one step procedure requiring only one hand and is easily executed.

* This instrument can be obtained from the E. B. Meyrowitz Surgical Instruments Co., 520 Fifth Avenue, New York 18, New York.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

October 8, 1951

DR. ADOLPH POSNER, *president*

OCULAR THERAPY

DR. ARTHUR DE VOE opened his discussion of ocular therapy with a brief survey of chemotherapy. He discussed the treatment of trachoma, inclusion blennorrhea, and meningococcus with the various sulfonamides, as well as with promizole, and gantrisin.

In the treatment of fungus infections, Dr. DeVoe mentioned the use of propion (five percent). This agent is also used for infections due to Pseudomonas.

Dr. DeVoe discussed the major considerations in the use of antibiotics in ocular infections: (1) The organism must be identified; (2) an adequate concentration of the drug must be used; (3) clinical tests of the drug must be made. He said that some strains of Pseudomonas are more susceptible to streptomycin than polymyxin. He also suggested that, if the infection does not respond within 72 hours to the antibiotic being used, it is well to change to another type.

Dr. DeVoe suggested that local use of the major antibiotics be avoided in ocular infections. He mentioned bacitracin (500 to 1,000 units/gm., gram-positive), neomycin (0.25 percent, gram-negative), and polymyxin (0.5 percent, gram-negative) as alternates to the major antibiotics. In acute conditions, he suggested use of the following drugs locally: penicillin (1,000 to 10,000 units/cc.), streptomycin (5,000 gamma), aureomycin (one percent, 5.0 mg./cc), chloramphenicol (one percent), and terramycin (0.5 percent).

For systemic use, maintenance of high blood levels is suggested. Dr. DeVoe mentioned the following antibiotics for systemic use: penicillin (300,000 units, twice a day), streptomycin (1.0 gm. maximum), aureomycin (0.5 gm., every six hours), terramycin (0.5 gm., every four hours), and chloramphenicol (0.5 gm., every six hours). Regarding chloramphenicol, Dr. DeVoe said that it probably penetrates best, is most stable, and results in less disturbances of the gastro-intestinal tract. He said that resistance to antibiotics may develop either due to the destruction of competitive organisms which may have been producing an antibiotic, or the antibiotic may actually be encouraging growth of some other organism.

Combinations of drugs, such as penicillin in combination with sulfa drugs, with streptomycin, with bacitracin, and with aureomycin were then discussed.

Antihistaminics, according to Dr. DeVoe, do not interfere with antibody formation, but diminish the undesirable effects of antibody-allergen union. They have, however, the side reaction of a local anesthetic, inhibit spreading of hyaluronidase, depress many tissues, may cause agranulocytosis, and may produce a hypnotic effect. Some have atropinelike effects and may dilate the pupils. In general, they have very little use in ocular disease.

Dr. DeVoe then discussed the use of such anticoagulants as heparin, dicumarol, tromexan, and the difficulties involved in their use. Agents for increasing clotting—vitamin K, vitamin C, calcium, and rutin—were discussed, as was the use of such vasodilators as amylnitrite, nitroglycerine, nicotinic acid, procaine, and priscoline in ocular disease.

Dr. DeVoe then mentioned the use of ACTH and cortisone in such ocular conditions as allergic dermatitis, keratitis, uveitis, sympathetic ophthalmia, and possibly optic

neuritis and retinitis. He discussed the dosage and systemic reactions of these drugs. He concluded his presentation with a brief survey of autonomic drugs and their use in ophthalmology.

Pathogenesis of glaucoma in relation to therapy

Dr. Adolph Posner said that since the etiology of glaucoma is relatively obscure, a rational approach to treatment should be based at least on a thorough understanding of the pathogenesis.

In acute congestive glaucoma, according to Goldmann, both the rate of outflow of aqueous and the pressure in the aqueous veins are elevated. The outflow pressure, however, is increased out of proportion to the pressure in the aqueous veins. Hence it must be assumed that there is increased resistance to the outflow of aqueous. In the narrow-angle type of acute glaucoma this resistance is probably produced by contact between the iris and the cornea. The permeability of the trabeculum itself is not impaired, at least not early in the course of the attack, before peripheral anterior synechias have formed.

Under these conditions, an iridectomy usually serves to cure the attack and to prevent recurrences. The iridectomy, moreover, does not have to be basal in order to accomplish a cure since the obstruction is at the entrance to the angle. An attempt to tear the iris out at its root is fraught with danger of complications such as hemorrhage and injury to the ciliary body, while any benefit derived from freeing the angle, or opening the trabeculum, is of questionable value.

After an attack of acute glaucoma has subsided, whether spontaneously or with miotics, the tension frequently drops temporarily to subnormal levels. This is an important finding since, in some instances, a diagnosis of glaucoma may be made merely on the basis of such a subnormal tension in the presence of a history suggestive of preceding attacks. This phenomenon can be explained by assuming that the outflow through the dilated aqueous veins continues at an accelerated rate even after the obstruction in the angle has been removed and after the resulting hypersecretion by the ciliary epithelium has subsided.

In acute glaucoma the sympathetic nervous system plays an important role, whether or not mechanical factors are present. This is particularly true during the stage of congestion and it explains why miotics, which usually succeed in aborting an attack in the precongestive stage, do not effect a cure so readily once congestion has set in.

On the other hand, experiences with the use of sympatholytic drugs—such as dihydro-ergocornine, -kyrptine, and -cristine—have shown that the tension can be lowered and pain relieved, at least temporarily, by an intravenous injection of these drugs, even if there can be no possible effect on the rate of outflow of aqueous, such as in the case of total posterior synechia, blood in the anterior chamber, or central retinal vein thrombosis. Other sympatholytic drugs such as dibenamine have given similar results. This effect may be ascribed to an inhibition of secretion of the aqueous by the ciliary body.

Acute glaucoma, usually with a minimum of congestive reaction, may occur in the absence of a narrow angle. There need not be any pathologic condition in the angle, such as obstruction of the trabeculum by particulate matter. In the intervals between attacks, the eye may be normal, even to provocative tests.

This is true of some cases of primary glaucoma and chronic congestive glaucoma. It is characteristic of one special group of cases, which are associated with the appearance of a few discrete keratic precipitates during the attack of ocular hypertension.

This type of glaucoma has been designated the "syndrome of glaucomatocyclitic crises" and was first presented by Posner and Schlossman before this society in May, 1947. In classifying this syndrome it is probably safest to regard it as an intermediate form between primary and secondary glaucoma

since the keratic precipitates by themselves cannot be considered as causing the glaucoma.

In chronic simple glaucoma there is an increased resistance to the outflow of aqueous. This resistance, according to Goldmann, is located in the trabeculum, although Ascher believes that it is situated in the ostia between Schlemm's canal and the aqueous veins.

In this form of glaucoma the rate of outflow of aqueous is normal, no matter how high the intraocular pressure may be. This fact seems to indicate that there is a regulatory mechanism present, which has for a purpose the maintenance of a constant rate of aqueous circulation.

This constancy in aqueous outflow has also been demonstrated experimentally by Bárány, who goes so far as to assert that the aqueous serves as a nutritive medium for certain avascular structures, such as the lens, and that an increased tension—within certain limits—may serve a useful purpose in maintaining the normal metabolism of these structures. It certainly is a well-known fact that cataract formation is frequently hastened by an operation which succeeds in reducing the ocular tension.

Considering, then, a moderate elevation of ocular tension to be a compensatory mechanism intended for the preservation of the normal functions of the lens, and, perhaps, other parts of the eye as well, it behooves us to be conservative in the treatment of early or mild glaucoma.

The criterion for surgery should be loss of fields, rather than elevation of tension. Several patients with mild glaucoma and elevated tension have been observed for periods of many years without noting any damage to the fields or to other ocular functions. In the majority of cases, it is true, the interference to the outflow, and therefore the increase in tension, takes on a progressive character.

For a time, miotics usually help in lowering the tension. The mechanism of action of these drugs is probably independent of their effect on the size of the pupil, although the latter is usually a good index of their therapeutic effectiveness. It is possible that they act through the hormonal or autonomic nervous system by either conserving, or substituting for, a chemical mediator such as acetylcholine, since Bloomfield has shown the aqueous of glaucomatous eyes to be deficient in this substance.

As the resistance to the outflow increases progressively, the miotics eventually prove ineffective in maintaining an adequate aqueous circulation. At some point, this disturbance of the normal function may call out a sudden vascular reaction in the ciliary body, resulting in an acute congestive attack. Lloyd of Oxford, England, has called attention to this terminal congestive phase in the course of simple glaucoma. In this phase, the obstruction is not at the entrance to the angle, as in narrow-angle glaucoma, but in the trabeculum.

Bernard Kronenberg,
Recording Secretary.

CHICAGO OPHTHALMOLOGICAL SOCIETY

November 19, 1951

DR. GLENWAY W. NETHERCUT, *president*

The Clinical Meeting was presented by the Departments of Ophthalmology, Presbyterian Hospital, and Chicago Medical School.

LOCALIZED EDEMA OF LID

DR. MARTHA RUBIN FOLK presented Mrs. S. G., aged 68 years, who presented herself on August 1, 1951, complaining of swelling of the left upper lid, ptosis, dryness of the skin, and itching, with an acute onset several months before. She had been under the care of several dermatologists and had undergone a complete physical examination. Laboratory findings were negative. On skin testing she

had been found mildly sensitive to various dusts and foods but no cosmetics. She had been treated with antihistaminics, cold cream, cold applications, and collyrium. Various antibiotic salves had been tried, resulting in a flareup of the condition. No treatment was effective and the symptoms continued with mild remissions and exacerbations.

Four months prior to onset of the condition her son died of acute leukemia. Since then she had been under great emotional stress characterized by frequent episodes of crying, during which she rubbed the eyes violently.

Vision was: O.U., (with a +0.75D. sph. = 20/20; add +2.5D. = J6 at 13 inches. There was edema and partial ptosis of the left upper lid with some drying of the skin and scaling. The palpebral conjunctiva was slightly injected and edematous. Corneal sensitivity was decreased. The pupils were equal and regular and reacted to light and accommodation. Tension was normal, fields normal, fundi normal.

The working diagnosis included localized edema with chronic dermatitis due to allergic or irritative causes or subacute glaucoma. Since the discs and fields were normal and there was no response to various provocative tests, glaucoma was eliminated. She was instructed to refrain from rubbing her eyes and was given topical suspension of 1:4 cortisone and vaseline for the skin. This seemed to aggravate the condition and was discontinued. She was placed on collyrium with ephedrine with symptomatic relief.

This probably represents an underlying component resulting in the original lesion, with a superimposed irritative component resulting from the patient's emotional condition.

Discussion. Dr. J. Vernal Cassady: This is an interesting case. There is pseudoptosis of the upper lid and edema which suggests herniation of orbital fat. If pressure is made on the eyes, the inner and outer part of the orbital fat protrudes on each side of the inferior oblique muscle. On pressure on the eyeball on each side of the superior oblique muscle the orbital fat protrudes. It has been suggested for relief of this condition that incisions be made through the skin and through the orbicularis, that the fat be cut out and the incisions closed, and that this would improve the patient's appearance.

Dr. Daniel Snydacker: I do not agree entirely with Dr. Cassady. Although he may be correct in the fact that there is a prolapse of orbital fat, it is also true that the skin is chronically inflamed. There may be both deep and superficial reasons for the bagginess of the eyelids. If dermatitis is present, one would have to be cautious in making incisions. In view of the clear history of emotional disturbance, psychiatric findings would be interesting, although Dr. Folk said there has been no results from psychiatric consultation.

Dr. Justin M. Donegan: The local use of cortisone in our hands in a case of proven allergic dermatitis has been of little value, whereas a few injections of ACTH, on the other hand, have given dramatic results.

Dr. J. M. Richardson: Might this be comparable to the type of condition found with hyperthyrotropic hormone disorder without exophthalmos?

Dr. Martha Rubin Folk (closing): The patient has been seen by two outstanding dermatologists who made numerous tests, and all were negative. As for the emotional factor, four or five times a day she goes into an hysterical state and cries and rubs her eyes, and the psychoanalyst said it would take many months of treatment to give her relief, and because of the expense she is unable to follow this through.

The basal metabolic rate was within normal limits. The medical department did not consider that a factor. The dermatologists are sure it is a localized edema. There is no evidence of exophthalmos and no other findings which would point to thyrotoxicosis.

Central retinal vein thromboses

Dr. J. Vernal Cassady reviewed 54 cases of central retinal vein thromboses that he had seen during the past 10 years. In his series of cases, central retinal vein thrombosis usually occurred in older persons with hypertension. The onset was sudden but often there was some premonitory visual disturbance. Emotional upsets, trauma, systemic diseases, or vasomotor disturbances may have been contributory factors in some cases.

Papilledema, retinal hemorrhages, exudates, and tortuous veins were found in the affected eyes. The profound loss of vision did not improve with reëstablishment of retinal circulation. Anticoagulant therapy, when used, did not appreciably influence either the retinal appearance or the recovery of function. There was no appreciable increase of vision either with or without anticoagulant therapy. Eighty percent of the patients remained blind.

Hemorrhagic glaucoma occurred in nine of the 54 cases, or 16 percent. In all of these patients, vision was lost. Six of the eight required enucleation to alleviate the stormy symptoms.

The prognosis of central retinal vein thrombosis is extremely grave and is directly related to the age of the patient and the degree of hypertension present. There is no effective treatment.

Discussion. Dr. Bertha A. Klien: Some of Dr. Cassady's observations in 54 cases of occlusion of the central retinal vein deserve emphasis.

There are several group studies similar to his in the literature, some of them concerned with a larger number of cases, but few mention consistently the condition of the vascular tree in the fellow eye. It was interesting, therefore, to hear Dr. Cassady state that in 17 of his cases there were obvious angiosclerotic findings in the other eye and that in two of these the venous occlusion was bilateral (12 percent).

It has been realized by most investigators for some time that a simple primary thrombosis of the central retinal vein does not exist. The occlusion of this vessel is the result of a combination of factors of which the most prominent appears to be a degenerative process in the intimal lining of the vein.

Pathologic study of such eyes reveals the surprising fact that these endothelial lesions are very similar, regardless of whether the occlusion occurred in an aged individual with simple senile angiosclerosis or in a younger patient with long-standing diabetes or hypertension. They consist of cushionlike proliferations of the endothelium, usually near the junction of the main branches, and, in some cases, of extensive detachment of the endothelial lining as in dissecting aneurysms.

In some cases, these almost standard intimal lesions are combined with prominent sclerotic findings in the central artery and the central connective tissue strand, which reduce the venous aperture still further by compression from the outside; while in others, the associated sclerotic changes of the neighboring structures are minimal and insignificant. In the latter cases the occlusive mechanism may be demonstrable as a direct complication of the intimal damage, as for instance, intramural thrombus formation.

More often the immediate cause of the occlusion is not apparent histologically, suggesting a combination of the endothelial lesions with stasis-producing disturbances in the hemodynamics of the retinal circulation, such as occur in various phases of hypertension or marasmus.

(Demonstration of cross-sections through the central vessels of eyes lost from occlusion of the central retinal vein in simple angiosclerosis, diabetes, and hypertension.)

It is not surprising that small benefit will be derived from treatment directed to influencing the blood-clotting mechanism in these cases of established occlusion, in which actual thrombus formation plays such a minor role or occurs in an inaccessible place.

If, however, similar endothelial lesions

are developing in the fellow eye, they may still be slight enough to permit repair if stress upon the vessel wall is reduced, and the danger of thrombus formation at the site of the damaged wall during periods of added venous stasis is lessened by anticoagulants. While these early intimal changes are not visible clinically, they are more likely to exist in an eye with visible angiosclerosis, and anticoagulant therapy should not be considered useless in such a patient even if it does not improve significantly the vision of the eye with the already accomplished venous occlusion.

As Dr. Cassady has emphasized, visual recovery does not depend so much upon reëstablishment of the venous circulation, as upon the extent of destruction of the macular retina. The damage to this area is usually extensive and largely irreversible in complete occlusion of the central vein, and may be almost as severe in occlusion of the superior temporal branch, which seems to be more than twice as frequent as that of the inferior branch. Early visual acuities, ranging from 20/20 to 20/70, which Dr. Cassady found in 20 percent of his cases, suggest partial occlusion of the central vein.

One claim made for anticoagulant therapy in central vein occlusion is the reduction in the incidence of secondary glaucoma, which Harold Falls, in the latest group study of such cases, quotes as nine percent in patients treated with anticoagulants as compared to 43 percent in cases not so treated. I would like to ask Dr. Cassady if some of the patients with secondary glaucoma in his series received this treatment.

Careful group studies of occlusive vascular disease of the retina, especially with reference to the more modern therapeutic armamentarium, should be encouraged, and it is hoped that others will follow his example. In the meantime, the most meticulous analysis and detailed long-range observation of individual cases will form the best basis for correct future statistical information.

Dr. James E. Lebensohn: Particularly interesting is the group of 10 percent of young people with incomplete thrombosis of the central vein and 20/20 to 20/30 vision.

I should like to report a similar case in a man, aged 25 years, whom I have had under observation for the past two years. He has now made a complete recovery and no longer are hemorrhages, exudates, or pigment visible. The present fundus picture shows a glial proliferation filling up the physiologic cup with adjacent neovascularization of the fine type described by Dr. Cassady. The patient was very introspective and noted the positive scotomas as spots scattered over the paper while he was reading. His fields were always normal except for a relative defect for blue. At the last interview all his subjective symptoms had completely disappeared.

Probably this case was due to a thrombus of the vasovasorum such as was illustrated by Dr. Klien and, when the reaction subsided, recovery ensued. The usual therapeutic measures aggravated rather than helped the situation.

Vasodilatation effected by niacin or by intravenous injection of typhoid vaccine caused more hemorrhages, as did also dicumarol. For a short time after administration of these agents some improvement would follow but then a new crop of hemorrhages would appear.

The condition was probably based on infection from the tonsils, as he had a history of recurrent tonsillitis and marked submaxillary lymphadenopathy. Removal of the tonsils was followed by an intensive course of chloromycetin, ascorbic acid, and rutin and these measures seemed to be the effective agents in his recovery.

Dr. William F. Moncreiff: In a large proportion of cases, the treatment of established (complete) thrombosis or obstruction of the central retinal vein is well characterized by the old proverb "locking the stable door after the horse is stolen." Accordingly, we can scarcely take issue with Dr. Cassady's conclusion that "there is no effective treatment" for this condition, despite the fact that his

conclusion would be much better supported in his own series had anticoagulant therapy, used more efficiently for longer periods of time and in all or most cases instead of in a small minority, proved to be equally ineffective.

The very hopelessness of therapy for eyes seriously damaged by an already accomplished obstruction of the central vein should stimulate us to give more attention to the possibilities of preventive anticoagulant therapy in early cases in which, by careful scrutiny of the retinal vascular tree, evidence is discovered of threatened obstruction of a central or branch vein.

There are two aspects of the problem of successful prevention:

1. Finding patients in the stage of premonitory but not complete obstruction, which requires that premonitory symptoms be taken seriously and, at least equally important, that a suitable fundus examination be made from time to time in all patients with vascular hypertension, diabetes, polycythemia vera, and any form of vascular or cardiovascular disease.

2. Efficient management of anticoagulant therapy. This subject, especially the matter of finding early cases, has been excellently studied and presented in a recent article by Dr. Klien.* The second aspect, efficient management of anticoagulant therapy, requires that the dosage of dicumarol or tromexan be safely and adequately regulated, for the ambulatory patient, so that the prothrombin level is kept at about 50 percent of normal or lower, for periods of months at a time.

This requires adequate supervision and laboratory estimation of prothrombin levels by the two-stage as well as the one-stage method at frequent intervals. Such laboratory facilities are expensive to establish and maintain, and require a considerable volume of work to be self-sustaining. Relatively few patients are located within easy access to the centers where such laboratory facilities are available, and still fewer are aware of their need for such therapy.

It is to be hoped that, as the latter problem is more fully solved, the increased demand will lead to more general establishment of these laboratory facilities.

Dr. J. Vernal Cassady (closing): I would like to thank Dr. Klien, Dr. Lebensohn, and Dr. Moncreiff for their discussion, and to say that Dr. Bertha Klien was the first to suggest the use of dicumarol in retinal vein thrombosis, in 1943. Most of us believe, as she does, that anticoagulant therapy should not be expected to be successful in occlusion of the central retinal veins resulting from either arteriosclerotic or inflammatory etiology. Dr. Klien pointed out and illustrated beautifully the endothelial changes present in the vessels. When not only this eye but the other eye is also involved, the patient might well be treated with anticoagulant therapy, either prophylactically or to prevent occlusion of the vessels in the other eye.

SCIENTIFIC PROGRAM

DR. GEORGE P. GUIBOR presented a paper on "Practical application of the neurology of the extraocular muscles."

Richard C. Gamble,
Secretary.

* Klien, Bertha A.: Prevention of retinal venous occlusion: With special reference to ambulatory dicumarol therapy. Am. J. Ophth., **33**:175 (Feb.) 1950.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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CAJAL

A CENTENARY APPRECIATION

With characteristic aptness Duke-Elder introduces his discussion of the retina in his *Textbook of Ophthalmology* with a portrait of Ramón y Cajal in acknowledgment of his epical revelations. The career of this pre-eminent neurohistologist was filled with unique contrasts. Penfield once remarked: "Cajal, Spain's first great scientist, is a man whose genius may be compared to that of Pasteur. He comes, like Pasteur, from the people and developed spontaneously on his native land." Don Santiago could be compared also to Weir Mitchell in the gift for popular writing, to Osler in his penchant for medical philosophy, and to his countryman, Gracian, in his flair for aphorisms.

Cajal's life, which began May 1, 1852, in Petilla, an obscure mountain village in northeastern Spain of about 50 homes, was at its parting the national symbol of intellectual distinction, commemorated with his effigy on banknotes and his portrait on a special postage issue. Though a leader in educational re-

SANTIAGO RAMÓN Y CAJAL

form and eventually Minister of Education, his own schooling had been marred till the age of 16 years by continuous recalcitrance, mischief, and inattention. His long life was astonishing, for immediately after finishing medical school in 1873 he was drafted for two years' service in Cuba from which he returned with the chills of malaria followed by the hemorrhages of tuberculosis. But illness, even when combined later with the infirmities of age, could not down his passion for medical science and in *The World at 80 —Memoirs of an Arteriosclerotic*, he described the onset and progress of his final malady; yet he entered medicine originally very much against his will. In a land of no opportunity presumably, especially in science, his success story, *Recollections of My Life*, outclasses the fantasies of Horatio Alger.

To Cajal's innate artistic talent, anatomy, and even more histology, made a special appeal. The "father of Spanish science" never studied abroad and in his formative years lacked personal contact with great investigators. Inspired by a view of some microscopic specimens that he chanced to see in 1877, Cajal achieved such a mastery of histologic technique through reading, ingenuity, and perseverance that he organized a course in normal and pathologic histology, published numerous articles (including one on the crystalline lens), and finally completed monumental treatises on these subjects that became world famous.

In 1887, the next pivotal experience occurred when sections of the brain impregnated with Golgi's stain were first shown to him. He immediately tested and perfected the stain, and applied the procedure zealously to the entire nervous system. He used young or fetal animals for the greater ease of following the course of nerve tracts and incidentally found that the stain thus gave more consistent results than when adult tissue was used. Prior to his investigations, the central nervous system was considered to be an intricate network of anastomosing fibers. In accordance with this conception the optic-nerve fibers were held to be continuous through all the retinal layers till their final termination as rods and cones. Cajal demonstrated that every nerve fiber is dependent on a nerve cell and that the nervous system is everywhere composed of anatomically discrete elements, whose free endings transmit by contact. This interpretation, dubbed the neuron doctrine by Waldeyer, was soon complemented by the idea of dynamic polarity of the neuron.

Cajal had an abiding interest in the retina because of its cerebral character, its definite function, and the stratified structure which facilitated analysis. He studied the retina extensively in chameleons, birds, rats, cats, and insects. He showed that in the vertebrate retina the centripetal pathway is composed of three separate links and contains at least two types of bipolar cells which he distinguished as the cone and the rod bipolar respectively. He assumed that in the fovea of

man as in that of chameleons and birds there is but one bipolar to each cone and that the one to one relationship continued between the foveal bipolars and the foveal ganglion cells. Cajal emphasized that this individual relationship of neurons in the fovea explained the greater acuity of central vision; and that the pattern of separate cone and rod pathways fitted well into the duplexity theory. Cajal also discovered and named the amacrine cells which he subdivided into various types. In the retina it was obvious that the dendrites conducted nervous impulses from the outside toward the cell body and that the axons were directed toward the central nervous cells. He found that this was true of all nerve cells and he called this orientation dynamic polarity.

Cajal continued his investigation of the neural pathway of vision with a timely study of the optic chiasma. The semidecussation of the human optic nerve, originally suggested by Wollaston, was being challenged by Michel who declared that the optic chiasma of man consisted exclusively of crossed fibers. Cajal proved that Michel's serial sections had been interpreted erroneously and by superior methods fully confirmed the traditional doctrine. After World War I Cajal studied in detail the retina and optic center of insects. He was fascinated by the efficiency and complexity of insect eyes and conjectured that the compound and simple eyes served photopic and scotopic vision respectively.

Ophthalmology is indebted to R. Greef who translated into German *The Retina of Vertebrates* in 1894 and Cajal's intervening contributions in 1898. In Greef's own monograph of 1899, Cajal's conception of the retina was given definitive presentation and since then the chief additional knowledge is concerned with the discovery of the midget bipolar and the midget ganglion cell. Cajal worked with analytical stains which for reasons still unknown select only a few nerve cells at a time out of the tissue mass. These techniques had been applied to the retina previously—the Golgi stain by Tartuferi, Ehrlich's vital methylene blue dye by Dogiel. Little was clarified by their research since both investigators clung to a doctrinaire adherence of the continuous network theory. Free from erroneous tradition, Cajal immediately came to the now-accepted interpretation of the elemental neuron structure of the entire nervous system. Spurred by the unremitting opposition of Golgi and his adherents, he contributed for 50 years a continuous stream of increasingly unassailable evidence—a record of unbroken achievement unparalleled in the history of medical investigation. Cajal almost single-handed doomed the network hypothesis to such oblivion that few today even know of this historic conflict. As the philosophic Cajal had anticipated: "The facts first associated with the name of one man end by becoming anonymous, lost forever in the ocean of universal science."

The Cajal monument in Retiro Park, Madrid. (Courtesy of Dr. Elmer Belt, Los Angeles, California.)

Foreign scientists ignored Cajal's flood of articles in the Spanish journals till his demonstrations before the Deutsche Anatomische Gesellschaft of Berlin in 1889 convinced them that a new star had arisen. The acclaim from abroad brought recognition at home and in 1892 Cajal, then aged 40 years, was promoted from the chair at Barcelona to Professor of Normal Histology and Pathological Anatomy at Madrid, a post which he occupied for 30 years. Nearly 100 honors

were bestowed upon him. Thanks to Sherrington's admiration he was asked to deliver the Croonian Lecture for 1894. In 1899 he participated in the decennial celebration of Clark University at Worcester, Massachusetts, where his lectures concluded with a fine tribute "to the great people of North America, . . . this daring race whose positive and practical intelligence . . . seems to be wonderfully endowed to triumph in the arena of scientific research." In 1904 came the Helmholtz Medal; and in 1906 he was awarded, with Golgi, the Nobel Prize at which time also the peace endeavors of our Theodore Roosevelt received recognition. Madrid honored him with the magnificent Institut Cajal and a beautiful symbolic monument in Retiro Park.

Besides several major treatises and about 265 scientific monographs, Cajal published books on color photography, popular science, science fiction, and literary miscellanea. The amazing Don Santiago was a witty talker, an indefatigable worker and a dynamic teacher, an admirable writer and an artist of distinction. The brilliant digest by Hilton of "Cajal's suggestions for scientific investigation" (*Scientific Monthly*, 36:225, 1933) is warmly recommended to every medical scientist, as it diffuses so vividly Cajal's inspiring personality. Ever mindful of his lowly origin, Cajal was politically a little to the left, but is that not where the heart is? The end of the master came on October 17, 1934, and his remains lie interred beside the body of his devoted wife in the Necropolis of Madrid. Modern ophthalmology owes much of its present brilliance to devotees of basic science such as Gauss, Young, Helmholtz, Gullstrand—and Cajal!

James E. Lebensohn.

ANTI-INFECTION AGENTS

Examples of dormant data

Metalnikoff, at the suggestion of Melchnikoff, attempted to find a substance that might destroy the waxy capsule on the tubercle baccillus and render the bacillus more vulnerable.

Metalnikoff found a small gray moth, the Galleria mellonella, that, in its life cycle, produces a caterpillar which subsists on honey. It robs the bees by invading the hives and boring holes through the honeycombs to get at the honey. After three years of study of the physiology and chemistry of this unusual caterpillar, Metalnikoff found that the digestive system and serum of this creature possessed an exceptional ability to digest wax. When the tubercle bacillus was introduced into a caterpillar's abdomen the waxy capsule melted away and the bacillus died. He repeated the tests in test tubes after extracting enough juice from the caterpillars. In vivo experiments in guinea pigs infected with tuberculosis were tried next. However, he required the extracts of thousands of caterpillars to heal a single guinea pig. He was able finally to treat a few successfully. Here his studies ended, since he never accumulated enough material to treat a human case. (Serge Metalnikoff: *L'infection Microbienne et L'immunité*. Paris, Masson & Cie, 1927.)

Recently two drug concerns have spent over a million dollars doing systematic, organized research developing a drug that appears to have considerable promise in therapy of tuberculosis; namely, the isonicotinic acid hydrazides. Actually this compound was described back in 1912 by H. Meyer and J. Nally in *Monatshefte der Chemie*, who never suspected the medical potentialities of their discovery.

Thus, the story of the sulfa drugs is repeated. In 1908, Gelmo described a new sulfur derivative of coal tar that remained an unused dye until in the 1930s the research of Domagk showed these sulfonamides to be potent bacteriostatic agents.

The penicillin story is similar. Accidentally discovered and thoroughly described by Fleming in 1929, it did not receive serious

consideration until the work of the Floreys over a decade later.

Scientific literature abounds in buried gems of scientific information. Must the unearthing of these dormant data await accidental discovery, new approaches, new theories, and years of repetitious research? Is there another way? Ophthalmologic literature is vast and is becoming larger with each passing year. The new review journals and review articles may be helpful to reseachers, but reviews of present-day literature will not uncover hidden knowledge of the past. Each investigator who accumulates new facts and postulates pregnant concepts must cover the past publications as exhaustively as his energy allows. The past may hold the answers to many of today's ophthalmologic questions. Irving H. Leopold.

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

The International Ophthalmological Council requested the national societies of different nations to suggest two subjects for discussion at the XVII International Congress of Ophthalmology which will take place in New York, September 12 through 17, 1954. At the meeting of the council on July 4th and 5th, the subjects chosen out of the numerous suggestions were *glaucoma* and *uveitis*. The openers of the discussion, also to be chosen by the council, will be announced in the near future.

Discussion on the two chosen topics will occupy the entire time of two assembly sessions. Other subjects will be discussed by a number of authors on a voluntary basis. A few have already been submitted to the secretary.

American ophthalmologists are responding generously to the call for financial contributions to the congress for preliminary expenses. An attractive membership certificate and pocket card are mailed to each contributor of 25 dollars as a reminder of the occasion when he can be host to his foreign colleagues and help make the Congress the success that is desired by us all.

William L. Benedict.

CORRESPONDENCE

AMERICAN JOURNAL OF OPHTHALMOLOGY

Editor,
American Journal of Ophthalmology:

Will you permit me to ask you a favor? Professor Kubik of Praha was a friend of mine in prewar time. After the war, I have tried to contact him but my efforts to reach him behind the iron curtain have been in vain. Finding his name in the May, 1951, number of the JOURNAL, I have thought that he might possibly have escaped to the western world. If you should by chance have his address, would you kindly send it to me?

I write further in the hope that it might interest you to know how much the JOURNAL is appreciated in countries far from the U.S.A. Since the end of the last war, I have been an interested reader of the JOURNAL, and my estimation for and gratitude to the editors is always increasing. I have not yet received a monthly number which did not contain papers of great interest to me. Collecting several papers on the same problem in the same monthly number is most inspiring—the result has been that the AMERICAN JOURNAL OF OPHTHALMOLOGY for a great part supplants the textbooks and monographs; and the JOURNAL always brings the most recent opinions.

(Signed) Per Riise,
Hamar, Norway.

BOOK REVIEWS

TEXTBOOK OF OPHTHALMOLOGY: VOLUME V, THE OCULAR ADNEXA. By Sir Stewart Duke-Elder, K.C.V.O., M.A., LL.D., D.Sc. (St. And.), Ph.D., (Lond.), M.D., F.R.C.S., Hon. D.Sc. (Northwestern), D.M. (Utrecht), F.R.C.S. (Edin.), F.A.C.S. St. Louis, C. V. Mosby Company, 1952. 1055 pages, 1181 illustrations, including 32 in color, index, references. Price: $22.50.

The eloquent pen of Stewart Duke-Elder continues to create for our essential use authoritative information on ophthalmology. In *Volume V* he brings us chapters on the ocular adnexa, which include the developmental anomalies of the eyelids, lashes, brows, skin of the lids, mobility of lids, lacrimal apparatus, and orbit. The next chapter covers the diseases of the lids, the most comprehensive of which is the discussion of the dermatoses, atrophies and degenerations, tumors and motor disorders. Then follows a chapter on diseases of the lacrimal apparatus; an excellent one on diseases of the orbit; and finally a most complete chapter on diseases of the para-orbital regions.

He follows his usual custom of introducing each section with a portrait and short biography of a world-important scientist whose contributions have played a significant part in the development of the knowledge concerning the pertinent subject. In this connection, it pleases us all to see that he has reproduced the photograph and familiar scrawled signature of our own Freddy Verhoeff who faces with calm imperturbability the chapter on diseases of the lacrimal apparatus. Duke-Elder points out that he was concerned with the difficulty of deciding just where to put Freddy, for "The reader of this textbook will have noticed that there is hardly a chapter on the physiology of vision wherein the name of Frederick Herman Verhoeff (1874-) does not appear and no chapter dealing with pathology in which his opinions do not demand weighty prominence."

It is a futile task to try to praise these volumes adequately. The lily cannot be gilded, and *Volume V* continues to carry on the theme of expert information in "glorious prose."

The volume on injuries, the sixth and final one promised us, is in preparation and then the author proposes to turn to the rather unhappy task of revising *Volume I* which is now a little out of date. May he be spared many more years of his life so that we may continue to benefit by his monumental genius, his quick wit, and his warm friendship.

Derrick Vail.

PATHOLOGY OF THE FETUS AND THE NEWBORN. By Edith L. Potter, M.D., Ph.D. Chicago, The Year Book Publishers, 1952. 554 pages, 601 figures, index, chapter references. Price: $19.00.

In this book the author gives a brief review of the early stages in development of the fetus and placenta in the human, and then presents the pathologic variances from the normal. She shows first the pathologic changes in the fetus in utero and then of the infant. As she states in the preface, the pathology is integrated with heredity, conception, development, intra-uterine and extra-uterine environment and behavior.

The first portion of the book deals with general causes of death and changes in the infant as a whole. Then the author takes up each portion or system (heart, gastrointestinal tract, skin) and describes pathologic details for each system, every one of which is covered.

The book is beautifully written with approximately 600 illustrations from material gathered from thousands of autopsies. Great care has been taken to describe and to show the pathologic changes. It is a book of exceptional value to pathologists, pediatricians, obstetricians, general practitioners, and med-

ical students. For such specialties as ophthalmology, otolaryngology, general surgery, neurology, and so forth there are only brief references.

There has been a great need for a book of this type and its appearance is especially timely because there is now a great deal of interest in fetal changes. This book meets that need adequately. Earl H. Merz.

PROCEEDINGS OF THE LONDON CONFERENCE ON OPTICAL INSTRUMENTS (1950). New York, John Wiley & Sons, 1952. 264 pages, illustrated, subject and name indexes. Price: $7.00.

Sir Thomas Merton, in his opening address, stressed how much civilization owed to optics and the periodicity of progress. Now "microscopy has come to life, not only with the development of phase contrast, but with the reflection microscope, interference microscopy and the development of microscopy in the ultra violet."

Within the past 15 years, American firms have introduced about 20 new optical glasses with refractive indices as high as n D 1.880. Undoubtedly many of these will eventually be used in spectacle lenses; indeed, Corning's n D 1.700, a marked improvement over dense barium crown, is now utilized exclusively in one firm's bifocals and trifocals. Plastic lenses have definite optical limitations, though in normal manufacturing operations an accuracy of 0.001 in the refractive index is readily attained. However, their thermal expansion is about 10 times that of glass, the refractive index decreasing by 0.00014 for each degree centigrade rise in temperature. Polystyrene, polycyclohexyl methacrylate, and polymethyl methacrylate are the only suitable optical materials. Polymethyl methacrylate is most affected by absorbed water, its refractive index being thereby increased by 0.002.

Of the 21 papers presented seven hail from Great Britain, six from United States, three from Sweden, two from France, and one each from Italy, Holland, and Switzerland. James E. Lebensohn.

TRANSACTIONS OF THE SOCIÉTÉS D'OPHTALMOLOGIE DE FRANCE 1951, No. 4, pp. 492-534.

The meeting of the Societe d'Ophtalmologie de Paris on April 12, 1951, and the meeting of the Société d'Ophtalmologie de l'Est à Strasbourg are discussed in this short volume.

G. Offret and Forest reported on a patient with spells of severe headaches of vascular origin, as proved by arteriography. G. Renard and Dinzard described their new equipment and method for detecting malingering of monocular amblyopia. Jourdy observed the rapid spread of a lymphosarcoma of the skull in an infant. The metastasis included both orbits. Jean Sedan saw a peculiar superficial keratitis in five contruction workers who handled a special kind of wood-polish and glue. Fellow workers of the same factory had occasional irritations of the skin of face and hands.

Thirteen papers were presented at the meeting of January 28th, in Strassbourg. A. Dollfuss discussed the importance of X-ray and radium treatment in cancerous growth and gave a clear summary on the possibilities of this type of treatment. He explained the technique and indication of the contact X-ray treatment as described by Chaoul which has been used in France with great success in superficial and localized lesions of the lids and globe since 1942.

Thomas, Cordier, Algan, and Mutel treated two children with bilateral retinoblastoma with X rays and radium without enucleation of the more affected eye. One child has stayed well ever since 1946. Although the other child improved and the tumor was apparently destroyed, a mass appeared in the blind eye after three years

and the optic nerve was found to be involved when the eye was finally enucleated. The child died 10 months later with widespread extension of the retinoblastoma to the base of the skull. No recurrence was visible in the other eye.

A. Gallois talked on corneal epithelial hyperplasias and Bowen's disease which he called hyperplastic corneal dyskeratosis with clumping cells. R. Levy and C. Heydecker had the opportunity to see a rubeosis iridis develop in patchy hemorrhages in the iris in an eye with thrombosis of the retinal vein and secondary glaucoma.

A. Lobstein and J. Muller discussed sympathetic ophthalmia and the importance of additional neurologic studies and spinal puncture for the prognosis of this disease. Thomas, Cordier, and Algan did a successful iridencleisis in a sympathetic ophthalmia when the tension could not be controlled otherwise. The diagnosis of sympathetic uveitis was made clinically and not verified pathologically.

A. Brini presented two patients with aneurysm of the internal carotid who showed mild papilledema, impairment of vision, central scotoma, and disturbance of color vision. The author emphasizes the importance of these ocular signs and symptoms for the diagnosis of an intracranial aneurysm. Heinz reported on the cataract extractions at the eye institute of the University at Innsbruck. He also demonstrated sections of the anterior segment of the globe.

Steiner discussed the possible toxic effects of PAS. In another paper he recommended aureomycin for herpes zoster and other superficial corneal lesions. C. Thomas, J. Cordier, B. Algan, and C. Vitte summarized their experience with subconjunctival injections of cortisone.

Appill noticed an increase in the sensitivity to glare with advancing age. Gerard, Fiel, and Grinault saw a squamous carcinoma develop in a fungus infection of the upper lid. They also consider the possibility of a secondary fungus infection in a primary carcinoma. X-ray treatment was used successfully.

Alice R. Deutsch.

GEOMETRY AND THE IMAGINATION. By D. Hilbert and S. Cohn-Vossen. Translated from the German by P. Nemenyi. New York, Chelsea Publishing Company, 1952. 357 pages, 330 diagrams, index. Price: $5.00.

Originally published in Germany in 1932 this is one of the many books, whose copyright is vested in the attorney general of the United States, which are now being translated for the American public. The firm sponsoring this particular opus specializes in mathematical works. With an abundance of diagrams the volume stresses the visual intuitive aspects of solid geometry in all its phases; and mentions tantalizingly their application to the various sciences and everyday experience. For example, a stone of any shape becomes increasingly similar to an ellipsoid as the water wears away at it; the conjugate foci of optics are compared to the two foci of an ellipse, and the cusp focus of a spherical mirror to the cusp of the epicycloid.

To the mathematically inclined this book will bring about a greater enjoyment of mathematics by making it easier to penetrate to the essence of the subject.

James E. Lebensohn.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Bembridge, B. A., Crawford, G. N. C., and Pirie, A. **Phase-contrast microscopy of the animal vitreous body.** Brit. J. Ophth. **36**:130-142, March, 1952.

Slitlamp examination has demonstrated that the vitreous is not a formless mass of gel but possesses a definite, organized structure. In this preliminary report the authors describe their studies of the physical and chemical structure of the vitreous bodies of the ox, pigeon and monkey. The eyes of these animals were examined by means of phase-contrast microscopy almost immediately after death, before postmortem changes could occur. For the photography a 90° positive phase-contrast microscope was used. Three definite and different structures were found within the vitreous body. Coarse branching fibers which attach the vitreous body to its base at the ciliary body were exposed by cutting a circular window through the sclera, choroid and retina, exposing the vitreous surface. A piece of the vitreous snipped off this region contains these coarse fibers which are attached to both the vitreous and the pigmented processes of the ciliary body. If the vitreous is pulled away, part of the pigmented processes of the ciliary body tears off with it. If a bit of vitreous body near the equator is cut off, the coarse fibers are not found; instead, a set of fine parallel fibers runs throughout the body of the vitreous. These fibers constitute the internal structure of the vitreous body. That the vitreous is contained within a hyaline membrane can be demonstrated by pushing the point of a knife through the surface of the body. The vitreous will herniate through the opening, whereas it had been contained before. This membrane, which has the appearance of crumpled cellophane, can be seen by the microscope in preparations made from the surface of the vitreous body. The vitreous body was subjected to the enzyme action of collagenase and trypsin; the former digested the fine fibers and the hyaline membrane, but not the coarse fibers, whereas trypsin digested the coarse fibers but not the fine ones. Morris Kaplan.

François, Jules. **Anatomical study of the retinal circulation.** Brit. J. Ophth. **36**: 37-40, Jan., 1952.

After injection of india ink into the central artery of the retina, the author found

that there is a superficial as well as a deep net of capillaries in the retina and the two networks anastomose freely. They are much denser at the posterior pole and become progressively less dense anteriorly. The retinal circulation is truly terminal; there is no direct connection between arterioles and venules. A junction is effected only through capillaries. The circulation of the superior temporal quadrant of the retina is not independent of the inferior, but a general, rich, capillary anastomosis exists between the two areas across the paramacular region. This suggests that arcuate scotomata are neuroscotomata and not angioscotomata. (7 figures) Morris Kaplan.

Sondermann, R. **Dynamics of development and the eye.** Arch. f. Ophth. **151**:380-383, 1951.

The purpose of this short paper is to emphasize the role of the developmental dynamics. In former studies the author had described the development of Schlemm's canal, the anterior chamber, the sphincter muscle and the ciliary processes as caused by an increase in resistance to venous backflow where the iridoscleral and lentociliary veins have to pass through the denser parts of the sclera. G. Dvorak-Theobald (1934) and P. E. Swindle (1937) confirmed these findings in well known investigations. The external muscles of the eye are also said to develop on the basis of obliterating orbital bloodvessels. Ernst Schmerl.

Wolff, Eugene. **Pacchionian-like bodies in the human canal of Schlemm.** Brit. J. Ophth. **36**:100-103, Feb., 1952.

There is a considerable misconception about the connective tissue content of the Pacchionian bodies and the structure of the inner wall of the canal of Schlemm. With Mallory's triple stain, a layer consisting of exceedingly fine fibrils was seen directly under the endothelium while deeper there were much coarser and much more loosely packed fibers. The fine fibers are the continuation of the arachnoid, the coarser ones of the subarachnoid trabeculae. A villus may be covered by a single layer of cells, but some form a multi-layered cap; beneath the endothelium is a layer of fine fibrils, while the core is formed of coarser subarachnoid trabeculae, or the whole villus may consist of the fine fibrils while the coarser ones run across the base. The inner wall of the canal of Schlemm may project into the lumen-like villus. It is usually lined by a single layer of cells, which are like those covering the Pacchionian body, but it may be in two or more layers, and may even form a multi-layered cap. The tissue directly under the endothelium consists of a recticulum of very fine fibrils; deeper down are trabeculae which resemble the subarachnoid trabeculae of a Pacchionian body. The trabeculae of the ligamentum, with their well-marked core of connective and elastic tissue, have the characteristics of scleral tissue and doubtlessly represent the dura through which the villus makes its way. There can be little doubt that Pacchionian-like bodies occur in the human canal of Schlemm. (4 photomicrographs) Orwyn H. Ellis.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Auricchio, G., and De Bernardinis, E. **The phosphate cycle in the retina and its biologic significance.** Ann. di ottal. e clin. ocul. **78**:53-60, Jan., 1952.

The retina can synthesize phosphated compounds of high energy content in vitro, in the course of its oxidation of glucose. The fact that acylphosphates are synthesized when sodium fluoride is present in a concentration sufficient to inhibit glycolysis, and likewise when the gluconate and the new 2-ketogluconate are

present, is new evidence that the retina can oxidize glucose directly, that is, without glycolysis as an intermediate step. The retina has a total acid-soluble phosphorus content but little less than that of the liver but with a different distribution of the component fractions: phosphorated compounds of high energy content are present in greater proportion, and the inorganic phosphorus is less. This difference is probably related to the high metabolic power of the retina as compared with the liver. Exposure to light tends to decrease the proportion of phosphorated compounds, perhaps because of their increased hydrolysis. (15 references)

Harry K. Messenger.

Baumann, A., and Cascio, G. **Post-operative hemorrhages in the anterior chamber in the light of present knowledge of the blood biology; with special reference to heparin activity of the blood plasma.** Arch. di ottal. **55**:633-648, Nov.-Dec., 1951.

Of 20 cases of post-operative hemorrhage examined with the toluidin blue method of Meneghini and Cervini, 11 had normal heparin activity and in 9 it was reduced but became normal after absorption of the blood. The authors believe that the heparin hypoactivity in the plasma is a transitory phenomenon closely related to disturbances of the blood coagulability, probably on the basis of a tissue and humoral allergy. John J. Stern.

Romero Robles, Eduardo. **The systems of three neurons of the retina.** Arch. Soc. oftal. hispano-am. **11**:1508-1512, Dec., 1951.

The author describes in detail and illustrates with drawings the marked structural and functional similarity between the construction of the tubes with three electrodes used in television and the structure of the nerve synapses, particularly those of the retina. Ray K. Daily.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Bornschein, H. **The threshold sensitivity to light of the human eye.** Arch. f. Ophth. **151**:446-475, 1951.

Each of 50 dark-adapted persons was studied 40 times, using light flashes of 0.5 seconds duration. The brightness thresholds were determined and statistically evaluated. The conclusions drawn from the statistical findings are that the individual deviations from the average are a distinctive quality of each person, not to be explained just as a phenomenon of quantum statistics. (217 references)

Ernst Schmerl.

Bottino, Carlo. **Clinical and experimental observations on the correction of high anisometropia with contact lenses.** Ann. di ottal. e clin. ocul. **78**:3-8, Jan., 1952.

The aniseikonia induced by ordinary spectacle lenses in high anisometropia can be easily measured with the Maggiore stereodiploscope and can be eliminated, with the establishment of comfortable binocular vision, by the use of contact lenses. These are recommended for patients with high anisometropia, unilateral aphakia, and high oblique astigmatism.

Harry K. Messenger.

Bottino, Carlo. **Asthenopia from extra-ocular causes, without errors of refraction.** Ann. di ottal. e clin. ocul. **78**:39-52, Jan., 1952.

Bottino found that asthenopia in patients with disease of the paranasal sinuses or with ovarian dysfunction is not relieved by glasses but usually disappears when the underlying condition is eliminated. He concludes that visual fatigue presupposes a neurovegetative instability in which axon reflexes along the trigemi-

nal nerve play a major part. (19 references) Harry K. Messenger.

Carreras Matas, Marcello. **Nocturnal myopia and its effect on the amplitude of accommodation.** Arch. Soc. oftal. hispano-am. **11**:1443-1489, Dec., 1951.

Thirty-one patients, of whom five were emmetropic, five presbyopic, five aphakic, five hyperopic, and one myopic were examined for refractive errors, dark adaptation and the degree of nocturnal myopia (determined by the method of Otero and Duran, using Ronchi's diafanometer). The graphically reported data show that the degree of nocturnal myopia in the young varies between 1.5 and 2 diopters. The author concludes that nocturnal myopia has a triple source; the spherical aberration caused by the pupillary dilatation in the dark accounts for no more than 0.25 of a diopter; the chromatic aberration in combination with Purkinje's phenomenon accounts for 0.4 of a diopter, and the increased curvature of the lens causes the rest. The crystalline lens appears to be the most important factor in the genesis of nocturnal myopia. No difference was noted in the degree of nocturnal myopia in hyperopes and myopes. It is lower in old people, and is lowest in aphakics, which shows that accommodation has a considerable influence on nocturnal myopia.
Ray K. Daily.

Hobbs, H. E. **Stereo-campimetry: a method for use with the Bjerrum screen.** Brit. J. Ophth. **36**:155-157, March, 1952.

Despite the fact that the accurate measurement of the visual fields is often of great value in diagnosis, the testing often becomes something of a guessing game between examiner and examinee. As failure is frequently due to faulty fixation, a method of stereoptic fixation is described by the author. The use of synoptophore bucket slides is incorporated into the chin rest; one half of the slide is projected on the screen while the non-fixing eye views the other half in a simple closed optical system. The method is not uniformly successful but is often an asset in difficult cases. Morris Kaplan.

Pallares, J. **Myopic anisometropia and the etiology of myopia.** Arch. Soc. oftal. hispano-Am. **11**:1513-1515, Dec., 1951.

Pallares finds support for the theory of a hereditary etiology of myopia in anisometropes with unilateral myopia. He has encountered unilateral myopia in families in which one parent is a high myope and the other a high hyperope. The separate development of each half of the human body makes it possible to inherit one eye from one parent and the other eye from the other parent. Ray K. Daily.

Roos, Wolfgang. **The image formed by corrected point-focal or wide-angle lenses.** Arch. f. Ophth. **151**:513-531, 1951.

A spherical lens is called a point-focal or wide-angle lens when the astigmatism of oblique pencils is abolished. In recent years lenses with weaker base curves have been manufactured, and it is claimed that these lenses meet the same requirements as the older point-focal lenses. The diameters of the newer lenses are increased up to 52 mm. A larger visual field is obtained and is useful as long as the marginal zones of the lens provide a satisfactory image. The newer lenses necessitate a re-definition of the image produced by point-focal lenses, the so-called punctual imagery. The older definition calls an imagery punctual when the astigmatism of oblique pencils is eliminated for a visual angle of 30° and an object at infinity. The larger diameters of the newer lenses make the following limitations necessary: a lens is considered a point-focal one, when the correction of the astigmatism of oblique pencils has reached an optimum for distances of from 25 cm. to infinity and for diameters up to 50 mm.,

and where the remaining astigmatism is practically not noticeable. The author determines the conditions necessary to obtain this optimum, gives the range of the corrected curves derived from trigonometric calculation, and shows that the results compare satisfactorily with the calculations used for the older point-focal lenses. Ernst Schmerl.

Sachs, Volkmar. **Light adaptation and the influence of privin upon sensitivity to glare.** Arch. f. Ophth. **151**:500-512, 1951.

The author investigated how much time an eye needed, when adapted to a definite brightness, in order to adjust to a sudden, glare-producing increase in brightness. He also examined the role of privin which is sometimes used to prevent over-sensitivity to light. Seven patients were repeatedly studied. The time of adaptation to light was found to be prolonged when the glare was unusually strong. Privin proved to be useful in diminishing the sensitivity to light and glare. (4 figures, 5 tables, references) Ernst Schmerl.

5

DIAGNOSIS AND THERAPY

Choyce, D. P. **Orbital implants.** Brit. J. Ophth. **36**:123-130, March, 1952.

Of 4,000 operations performed at Moorfields hospital in 1950, 200 were enucleations of an eye. The purpose of this paper is a study of the survival rate of orbital implants in these patients. Ninety-eight percent of the cases were studied after 12 months; they represented various types of integrated implants inserted by many surgeons using various techniques. In all the rectus muscles were attached directly to a gold ring or to the tantalum mesh, while Tenon's capsule and the conjunctiva were separately sutured. The immediate results were excellent in that appearance and motility were satisfactory. However, after two years almost half the implants had been extruded or had been removed, and it is probable that half of the others will soon be lost. A state of chronic infection was the cause of the loss in most cases. The junction between the conjunctiva and the plastic implant seems unable to withstand bacterial invasion. The fate of the implant was not affected by the age of the patient, reason for enucleation or ability of the surgeon. The author suggests that the use of unburied orbital implants be discontinued and that ophthalmic surgeons continue to seek a solution to this problem.

Morris Kaplan.

Couadau, A., and Campan. **Curare in ocular surgery. Observations on 100 cases of cataract extraction.** Arch. d'opht. **12**:48-57, 1952.

The authors report on 100 cases of cataract extraction in which curare was employed. They analyze the cases in detail according to the effect of the curare on the orbicularis and the extraocular muscles, on the intraocular tension, and on the vitreous body. Disturbances in swallowing were noted in 20 cases as complications of curare therapy. This effect, though benign, is disturbing to the patient and can be relieved by the preoperative administration of atropine. Diminution in the amplitude of respiration, although unimportant, annoys the patient. Fifteen patients showed a postoperative hypotonus of the arms and 18 a hypotonus of the legs, only perceptible in the standing position. The vitreous was not lost in a single case, neither did the patient cough or sneeze during the operation. The authors conclude that curare is of real benefit in controlling the major complications of intraocular surgery. P. Thygeson.

Dellaporta, A. **Experimental shortening of the bulbus in animals.** Arch. f. Ophth. **151**:403-445, 1951.

The author uses dogs for his surgical procedures. He finds that the eyeball can

be shortened by several millimeters. A folding of the retina and choroid may take place and usually disappears within two weeks. Where the retina does not become attached again within three weeks, degenerative changes develop. The literature is discussed in detail. (18 figures, 40 references) Ernst Schmerl.

Dorello, Ugo. **The value of Mester's reaction in ophthalmology.** Arch. di ottal. **55**:649-660, Nov.-Dec., 1951.

Mester's reaction consists of the intradermal injection of five doses (1 mg. each) of sodium salicylate at one time. Thirty or 60 minutes after the injection a marked leucopenia can be detected in persons with rheumatoid arthritis. The author tested this method in 20 cases of inflammation of the anterior segment (iridocyclitis, episcleritis) and in ten normal subjects concluded and that it is of no value in recognizing the etiology of these eye diseases. John J. Stern.

Kleckner, J. F. **A test for monocular malingering.** U.S. Armed Forces M. J. **2**: 1911-1914, Dec., 1951.

An easily constructed, simple wooden apparatus, devised to ascertain the presence of reading ability in both eyes and the degree of acuity present, is described and measurements for its construction are given. The apparatus is intended for malingerers and amblyopes and does not rule out true hysteria. F. M. Crage.

Lepri, Giuseppe. **First clinical results with local cortisone in ophthalmology.** Arch. di ottal. **55**:607-622, Nov.-Dec., 1951.

Seventy-nine patients with disease of the anterior segment or the ocular adnexa showed the now well-known beneficial influence of local cortisone therapy.

John J. Stern.

Lepri, G., and Sicca, G. T. **The penetration of locally administered terramycin into the aqueous.** Arch. di ottal. **55**:383-396, July-August, 1951.

Titration of terramycin in the aqueous by biologic assay showed that this substance did not penetrate into the aqueous of rabbits after instillation of a buffered solution, or of an ointment into the conjunctival sac, with or without previous artificial abrasion of the cornea. After subconjunctival administration, a low concentration of terramycin was obtained in the aqueous. The highest level was reached after one to one and one half hours; after two hours, no trace was found. John J. Stern.

Pierret, R. **Observations on mineral water therapy in ophthalmology.** Arch. d'opht. **12**:42-47, 1952.

Eye diseases have been treated in mineral springs health resorts since antiquity but no comprehensive studies of the effect of such "cures" have been made. Pierret has studied the effect of the waters of the thermal station of La Bourboule on toxic, allergic, and infectious eye diseases. Allergic diseases, including vernal catarrh, are reported as doing well at this station; radioactive and arsenical mineral water is added to a high meat diet with benefit. The tonic effects of the waters of this station are of value also in toxic affections, particularly those complicating diabetes. In ocular infections there is an especially favorable effect in blepharitis and blepharoconjunctivitis due to staphylococci. This is attributed to the hypoglycemia-producing effect of the waters.

P. Thygeson.

Rama, G. **Cortisone and corticotrope substances in ophthalmology.** Arch. di ottal. **55**:461-470, July-August, 1951.

Local and parenteral administration of cortisone, an ultrafiltrable polypeptide fraction of the corticotropic hormone, and Δ_5-pregnenolone were used in a number of

eye conditions. The author was convinced that the best results were obtained in chorioretinitis and iridocyclitis. One case of episcleritis and one of follicular conjunctivitis responded moderately well, two cases of glaucoma, two of exudative choroiditis and one of rodent ulcer did not respond to the treatment.

John J. Stern.

Sgrosso, S. **A new apparatus for the measurement of the flicker fusion frequency.** Arch. di ottal. **55**:471-476, July-August, 1951.

A device for the measurement of the flicker fusion frequency is described which consists of a perimeter half-arc, a light source (10 lux) arranged in the horizontal axis and a rotating perforated disc. A mirror allows the light to be projected on any point of the perimeter arc from the extreme periphery to the region immediately adjacent to the fovea, and also to test two different points of the field simultaneously. John J. Stern.

6

OCULAR MOTILITY

Braley, Alson E. **Management of convergent strabismus esotropia.** J. Iowa M. Soc. **42**:106-110, March, 1952.

One of the major causes for failure is insufficient cooperation between physician and parents, as the study of these patients requires repeated visits to the physician. Treatment must correct refractive errors, amblyopia, and abnormal retinal correspondence, and the esotropia must be treated by surgery. The decision for surgical correction depends on the age of the patient, effect of glasses on the squint, the degree of the deviation, the activity of the rectus muscles during rotations, and the appearance of the esotropia under anesthesia. Generally, the straighter eyes appear under anesthesia the less resection and recession is needed. In a young child when esotropia is still present under anesthesia, recession of the medial rectus up to 5 mm. and resection of the lateral rectus up to 10 or 12 mm. may be done. A table shows the technique and the author's results in 44 cases.

Francis M. Crage.

7

CONJUNCTIVA, CORNEA, SCLERA

Bagolini, B. **Dendritic keratitis of corneal grafts.** Boll. d'ocul. **31**:41-45, Jan., 1952.

In a cornea with a penetrating transplant, and in one with a lamellar transplant, herpetic keratitis developed less than two years after the keratoplasty. The host cornea remained unaffected in both cases, only the flaps were affected. The author assumes that the transplant may have offered less resistance to the virus than the surrounding cornea.

K. W. Ascher.

Chiniara, J. **An unusual form of keratoconjunctivitis.** Arch. d'opht. **12**:61-65, 1952.

Chiniara describes 27 cases of an acute keratoconjunctivitis which he observed over a seven-year period. The disease was characterized at onset by a conjunctival hyperemia and peripheral corneal infiltration, and later by ulceration and vascularization. It was self-limited, usually within 12 days, and the only residual was a small corneal cicatrix at the site of the ulcer. In his discussion, the author differentiates the disease from phlyctenular keratoconjunctivitis, herpes of the cornea, syndrome of the nasal nerve, corneal rosacea, epidemic keratoconjunctivitis, and allergic conjunctivitis. No bacteriological or cytological examinations of the conjunctival exudates are reported. P. Thygeson.

Franceschetti, A., and Forni, S. **Hereditary crystalline corneal degeneration and its relation to the heredofamilial corneal**

dystrophies. Boll. d'ocul. **31**:3-20, Jan., 1952.

The case of a 6-year-old girl with crystalline degeneration of the cornea called to the authors' attention the necessity of a new classification of the heredofamilial corneal degenerations. They distinguish the following groups: parenchymatous degenerations, degenerations of the limiting membranes, combined forms, and corneal degenerations in general disease. The clinical course, histology and mode of transmission of most of these different disease entities are discussed. (13 figures, 45 references) K. W. Ascher.

Goar, E. L., and Smith, L. S. **Rheumatic nodules of the eye.** J.A.M.A. **148**:889-892, March 15, 1952.

A diagnosis of rheumatic nodules of the sclera was made after pathologic examination in two cases. In one patient, enucleation was performed after a perforating pneumococcus corneal ulcer and a much thickened scleritic nodule. In the second patient, uveitis and perforation of a scleritic nodule in a blind eye were followed by enucleation. In neither case was the diagnosis of the ocular disease considered as part of the rheumatoid state. Intercurrent disease so obscured the original disease that not until a microscopic examination was made was its true nature recognized. Except for minor variations, the histologic lesions about the joints were similar to those found in the scleritic nodules. Since certain types of diseases of the eye are a part of the rheumatoid state, it is considered of great importance that the relationship be determined early in the disease, especially since such good results are obtained with adrenal steroids and corticotropin. (9 figures)

F. M. Crage.

Lopez Enriquez, M. **Biomicroscopy of the eye in polarized light.** Arch. Soc. oftal. hispano-am. **11**:1516-1518, Dec., 1951.

Lopez calls attention to the almost forgotten work of Koeppe on biomicroscopy of the cornea in polarized light. He believes that the corneal architecture revealed by this method of examination must not be neglected when inserting a corneal graft. It may be of importance to the visual and tectonic result to have the graft correctly oriented in relation to the fascicular pattern which is revealed by polarized light. (4 figures)

Ray K. Daily.

Renard, G., Lelievre, A., and Naneix, G. **Cicatrization of wounds of the sclera.** Arch. d'opht. **12**:5-18, 1952.

The authors comment on the problem of wounds of the sclera and note that the majority of ophthalmologists practice a simple conjunctival covering of the wound without scleral sutures. They studied the repair by careful conjunctival coverage of wounds in the rabbit sclera. The wounds were made by various methods, including simple Graefe incision and resections of sclera with or without perforation of the underlying choroid and retina. In addition to scleral sutures they employed grafts of 1. fascia lata preserved in alcohol, 2. cartilage preserved in alcohol, 3. sclera preserved by refrigeration in penicillin solution, and 4. fresh cartilage. Their studies included histopathologic documentation of the wounds at various stages. They conclude that after coverage with conjunctiva alone, good cicatrization takes place, but that if the choroid and retina have been punctured and vitreous lost, the fibroblastic tissue rapidly invades the vitreous cavity. For this reason they stress the importance of scleral sutures whenevre possible. Of the grafts employed, only fresh cartilage proved to be of value. In one patient with a typical scleromalacia perforans, a fresh cartilage graft from the patient's ear was used to cover the perforation. The result was good.

P. Thygeson.

Salvi, G. L. **Clinical study of the therapeutic action of cortisone and ACTH on vernal conjunctivitis.** Boll d'ocul. **31**:46-58, Jan., 1952.

Five tables review the histories of 25 patients with vernal conjunctivitis; 15 were treated with ACTH, 10 by topical application of cortisone. The age, type of disease, treatment, duration of observation, and results are recorded. Topically used, cortisone was more effective than ACTH given systemically; six patients previously treated with ACTH improved only after cortisone was administered topically. Recurrences are to be expected after cessation of cortisone treatment. (3 tables, 20 references) K. W. Ascher.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

France, R., Buchanan, R. N., Wilson, M. W., and Sheldon, M. B., Jr. **Relapsing iritis with recurrent ulcers of the mouth and genitalia (Behcet's syndrome).** Medicine **30**:335-356, Dec., 1951.

The case reported is one of 33 in the literature described as Behçet's syndrome. The patient, a 29-year-old white man had had biopsies taken from lesions on the scrotum and knee joint. Enucleation of both his blind eyes had been performed approximately six years after the onset of the disease. The blindness was the result of a hemorrhagic and exudative uveitis and there had been severe retro-orbital headaches. In a review of the literature it is pointed out that no treatment has been found to stop the progress of this malignant relapsing iridocyclitis. Twenty-five percent of the cases reviewed showed a thrombophlebitis, and biopsies in this case showed inflammatory lesions in the small veins. (8 figures, 1 table)

H. C. Weinberg.

Hilding, A. C. **Syndrome of joint and cartilaginous pathologic changes with destructive iridocyclitis.** Arch. Int. Med. **89**: 445-453, 1952.

An elderly woman with chronic rheumatoid arthritis developed degenerative changes of the cartilages with abnormal mobility of the joints, multiple dislocations, deformities of the cartilages of the nose and ears, and a plastic iridocyclitis with cataract formation and hypotony. Extraction of the cataract was of no value in restoring vision.

A brief description of the uveal circulation and the formation of aqueous humor is given. The uveal tract may easily be involved in inflammatory processes from various causes, infectious, allergic, or toxic and these general systemic diseases may damage the capillary walls of the uvea and affect permeability and the production of aqueous.

The association of eye symptoms in several diseases that involve the joints is quoted from an article by Godtfredsen, and the similarity of the eye and joint involvement is shown pathogenically and histologically. Godtfredsen stated that the blood-aqueous and blood-synovial barriers are similar, and that the ciliary processes of the eye and the synovia of the joints are both susceptible to streptococci.

The author found no other recorded case similar to the one he describes in the literature. He believes his patient's disease is related to rheumatoid arthritis, but differs in several respects. The cartilaginous changes are unexplained. (2 tables, 4 figures) James W. Brennan.

Marsico, Vincenzo. **Tissue therapy for sympathetic ophthalmia.** Arch. di ottal. **55**:675-688, Nov.-Dec., 1951.

Three cases of sympathetic ophthalmia were treated with Filatov's method, with excellent results. John J. Stern.

Silfverskiöld, B. P. **Recurrent uveitis (Behçet's syndrome) and encephalomyelomeningitis.** Acta Psychiat et Neurol. **26**:443-453, 1951.

The author reports three patients who had neurologic symptoms. All three had recurrent spastic paresis of the extremities, chiefly in the form of paraplegia; one also had speech and swallowing difficulty, a second paresis of the sixth and seventh cranial nerves, and the third had an intention tremor. The neurologic disturbance was preceded by uveoretinitis with markedly diminished vision. Recurrent hyperpyrexia, pleocytosis in the spinal fluid, and a great increase in the sedimentation rate were likewise present. The condition must be differentiated from undulant fever and benign lymphogranulomatosis. A virus is supposed to be the causative agent, but in one of the author's cases a gonorrheal infection was present and in another there was an infection in the tonsil. F. M. Crage.

9

GLAUCOMA AND OCULAR TENSION

van Beuningen, E. G. A. **The optical density of the corneo-scleral trabeculum in the normal and in the eye with primary glaucoma.** Arch. f. Ophth. **151**:532-540, 1951.

The author combined gonioscopic procedures with the use of Roenne's colloidometer to determine the optical density of the tissues which occupy the angle of the anterior chamber and examined 280 normal and 273 glaucomatous eyes. In primary glaucoma, a slightly higher density was found than in normal eyes. However, it seems to be doubtful whether this difference can be considered significant. Ernst Schmerl.

van Beuningen, E. G. A. **Elliot's trephine operation, cyclodialysis and iridectomy, and their indications, based upon gonioscopic observations.** Arch. f. Ophth. **151**:541-550, 1951.

The author studied a large number of glaucomatous eyes and concluded that: 1. the Heine-Blaskowicz cyclodialysis is indicated in wide angle glaucoma; 2. a preliminary peripheral iridectomy should be performed where adhesions block parts of the angle of the anterior chamber and a cyclodialysis should follow; 3. Elliot's trephine operation is preferable where the adhesions reach higher degrees; and 4. the dangers of any surgical procedure increase with advanced iridocorneal adhesion and occlusion of the angle of the anterior chamber. Where a malignant glaucoma is suspected, conservative treatment is indicated. Ernst Schmerl.

10

CRYSTALLINE LENS

Arruga, Alfredo. **The problem of inclusion of the lens in Ridley's operation.** Arch. Soc. oftal. hispano-am. **11**:1490-1507, Dec., 1951.

Ridley's extracapsular cataract operation, with inclusion of a plastic lens, was performed by the author in nine cases. He suggests that this operation should not be attempted when there is an atrophic or rigid iris. In one case of this type the plastic lens was dislocated into the vitreous, and could not be extracted. The author uses a retrobulbar injection, corneal keratotomy and closes the wound with silk sutures on Grieshaber needles. The anterior capsule was removed with capsule forceps, the nucleus expressed, and the cortex thoroughly removed by irrigation. The plastic lens was inserted with forceps. It is important that the lens be perfectly centered. Postoperatively there is pigmentation on the surface of the lens and the formation of synechia. Vision improves after some months as the pigment on the lens surface is absorbed. (15 figures, 15 references) Ray K. Daily.

Pau, Hans. **Transitory changes of refraction and cataract.** Arch. f. Ophth. **151**: 560-564, 1951.

From studies reported in an earlier paper, the author concludes that swelling of the lens and increase in weight make the lens more spherical, thus increasing its power of refraction. Dehydration of the lens and loss of weight flatten its surface and decrease its power of refraction.

Ernst Schmerl.

Pau, Hans. **Mechanisms involved in accommodation.** Arch. f. Ophth. **151**:565-573, 1951.

The author studied the forces involved in the changes of the shape of the lens during accommodation. He used lens-zonula--ciliary-body preparations and found that a contraction of the ciliary body corresponding to the pull of a weight of 1 gram in one direction flattens the lens in this direction by 1 diopter. The distensibility of the tissues increases in the following order: anterior capsule, posterior capsule, zonular fibers. The colloid osmotic pressure of the lens substance of calves approximates 1,000 mm. of water. This pressure counteracts the pull exercised upon the lens during the activity of the ciliary body.

Ernst Schmerl.

Renard, G., and Laporte, P. **Congenital cataracts with crystal formation.** Arch. d'opht. **11**:739-753, 1951.

A child of 8 years had an unusual congenital cataract characterized by masses of crystals in the axial portions of the two lenses. In the differential diagnosis the two forms of congenital cataract with crystal formation, the "spiesskatarakt" of Vogt and the coralliform cataract, are considered, and the reported case classified as belonging to the latter type. Attention is called to the familial character of the disease and to the fact that the nature of the crystals is still undetermined. The rarity of congenital cataract with crystal formation (only 20 reported cases) is noted.

P. Thygeson.

Ridley, Harold. **Intra-ocular acrylic lenses.** Brit. J. Ophth. **36**:113-122, March, 1952.

There are many disadvantages to aphakia and the replacement of the removed lens is the ideal solution. The use of a human lens is still impossible; an artificial lens has been used in 27 eyes since 1949 with an amazing degree of success. The choice of material lay between glass and one of the acrylic substances, both being inert and non-irritating. The plastic had the overwhelming advantages of being half the weight of glass. A type of plexiglass was selected whose refractive index is 1.49 and whose specific gravity is 1.19. It is easily sterilized by immersion in proper solutions. A standard lens is used in all cases although it is foreseen that eventually lenses will be individually made for each separate eye. The design of this artificial lens presented many problems. The specifications which were finally selected continue to be used. The lens is made of polymerized polymethyl methacrylate; it measures 8.35 mm. in diameter and 2.40 mm. in thickness; the anterior radius of curvature is 17.8 mm. and the posterior 10.7 mm. A. peripheral groove on each side permits firm grasping by forceps. The refractive power is +74D in air and +24D in aqueous humor.

A special grasping forceps and a special sterilizing container are designed for use; the container holds two lenses which are placed with the anterior side forward; 1-percent cetrimide solution is used.

At present extracapsular extraction is recommended, although in two cases intracapsular extraction was used successfully. The pupil is dilated with homatropine only and local anesthesia is used without retrobulbar injection. Two half depth corneoscleral sutures are put in place and an ordinary extracapsular extraction is done. All of the cortical ma-

terial must be irrigated from the anterior chamber. The acrylic lens is grasped with the forceps and the section opened with an iris repositor; the lens is guided into and through the pupil to rest below the lower half of the iris and then slipped under the upper iris with the aid of an iris hook. Final centering is done by external pressure on the cornea by iris repositors. A small peripheral iridotomy is then done and the anterior chamber again irrigated after which the two sutures are secured. Penicillin is instilled and both eyes are covered for 48 hours. Miotics are not considered necessary. The patient gets out of bed on the third or fourth day and the sutures are removed on the eighth day. There seems to be little danger that the lens will become dislocated once it is properly inserted; it has not occurred in this series. Three complications occurred: in one eye, the posterior capsule became thickened and wrinkled and necessitated a capsulotomy which was done posteriorly through the sclera; in a second, iris prolapse occurred which healed well, and in a third the anterior chamber collapsed and glaucoma developed. A mild serous iritis with transient adhesions between the pupillary margin and the lens usually follows but this heals promptly. In all cases binocular vision has resulted and in many cases the spectacles used before cataract development could be used again. It is not impossible that this technique will eventually make all previous cataract extraction techniques obsolescent.

Morris Kaplan.

Sbordone, Girolamo. **Clinical notes and pathogenetic considerations on lenticular changes after keratitis with hypopyon.** Ann. di ottal. e clin. ocul. **78**:17-24, Jan., 1952.

Sbordone describes and discusses the lenticular changes observed in ten patients. The principal pathogenetic factors are thought to be the toxic content of the anterior chamber during the active period of the keratitic hypopyon, alterations in the permeability of the lens capsule, and modifications of the metabolism of the lens. (13 references)

Harry K. Messenger.

11

RETINA AND VITREOUS

Jefferson, M., and Cloake, P. C. P. **Nervous manifestations in Eales's disease.** Brit. J. Ophth. **36**:143-150, March, 1952.

In the original description of Eales's disease, the recurrent retinal hemorrhage was ascribed to a vasomotor neurosis in which a vasoconstriction of the abdominal vessels caused a compensatory dilatation of the vessels of the head and resulted in intraocular bleeding. The disease is a clinical entity and the bleeding is recurrent. There have been two main theories as to its etiology, both of which are probably erroneous. For many years it was believed to be a manifestation of tuberculosis, but repeated histologic studies have failed to bear this out. That it was a manifestation of Buerger's disease in the vessels of the eyes seems unlikely. Studies in numerous cases of Buerger's disease have shown no involvement of the eyes, nor was there peripheral involvement of the vessels in any of the cases of Eales's disease.

Histologic studies show the lesion to be one of patchy cellular infiltration of the walls of the blood vessels, mostly the peripheral veins of the retina. The infiltration is lymphocytic and is found also in the optic nerve. It has been suggested that the infiltration might be an expression of allergic endophlebitis caused by a large variety of allergens. Neurologic complications have been reported from time to time but none were present in the six typical cases of Eales's disease reported by the authors, nor was there

evidence of either Buerger's disease or tuberculosis. Morris Kaplan.

Kahan, A., and Sipos, M. **The structure of the macula. Studies based on examinations of patients showing a tapeto-retinal degeneration.** Arch. f. Ophth. **151**: 476-499, 1951.

Nine members of four families were examined for sense of brightness for different wave lengths and color vision, their fields were studied for color, and light adaptation was tested. In three cases of the type first described by Best, the morphologic changes of the pericentral zone were found to be associated with functional damage to the whole retina. A total achromatopsia seemed to exist. The cases of the type first described by Stargard, showed damage of the blue perceptors of the periphery and pericentral area. Green perceptors seem to be damaged in the more peripheral parts of the retina. The healthy relatives of the examined patients showed some disturbances of light adaptation, which seemed to the authors to be suggestive for their quality as conductors. (12 figures, 82 references)

Ernst Schmerl.

Sbordone, G. **The photomotor reflex in detachment of the retina.** Arch. di ottal. **55**:439-460, July-August, 1951.

In analyzing 15 cases of detachment of the retina with Sander's differential pupilloscope, it was found that the differential luminous threshold which provokes the photomotor reflex is more or less markedly raised according to the site, the bulging and the extent of the detachment. In nine subjects with surgically repaired detachment, the threshold was either normal or slightly raised depending upon the extent and the type of detachment, and the time that had elapsed between the onset of the disease and the operation.

John J. Stern.

12

OPTIC NERVE AND CHIASM

Maione, Mario. **Optic atrophy from minor trauma in the orbitofrontolparietal region.** Ann di ottal. e clin. ocul. **78**:33-38, Jan., 1952.

Maione briefly describes five cases of rapidly progressive homolateral optic atrophy which began about 10 days after a contusion or laceration of the soft tissues surrounding the orbit, that is, in the region supplied by the ophthalmic nerve. These cases support the old concept of a reflex amaurosis, especially in view of the close relation between the trigeminal nerve and autonomic ganglia such as the sphenopalatine, ciliary, and otic. (5 references) Harry K. Messenger.

Mecca, Mario. **A tumor of the optic nerve and Recklinghausen's disease.** Ann. di ottal. e clin. ocul. **78**:61-75, Jan., 1952.

Mecca reports a case of an optic nerve tumor, probably an astrocytoma, removed from a 16-year-old boy with Recklinghausen's disease. (10 figures, 36 references) Harry K. Messenger.

14

EYEBALL, ORBIT, SINUSES

Culler, A. M., Guyton, J. S., Hughes, W. L., Cutler, N. L., Troutman, R. C., and Stone, W., Jr. **Symposium: Orbital implants after enucleation.** Tr. Am. Acad. Ophth. **56**:17-41, Jan.-Feb., 1952.

Culler, Arthur M. **Basic principles of anatomy and physiology of the orbit and relation to implant surgery.** pp. 17-20.

Accurate replacement of volume and placing the posterior pole of the implant in the position of the posterior pole of the globe minimize tissue migration. Strain on the attachments of the muscles to the implant is reduced if they are inserted anterior to the center of rotation of the im-

plant and prosthesis. Friction and pinching of tissues are reduced if there is accurate coaptation between the implant and the prosthesis. Tantalum mesh promotes fibroplastic proliferation and should be utilized only where strong adhesions are desired. Suturing in tantalum mesh promotes fragmentation. The conjunctiva becomes thickened, tough and inelastic unless it is utilized under some tension or pressure.

Guyton, Jack S. **Procedures and physiologic factors.** pp. 21-24.

Buried "motility" implants are probably superior to spheres because they are likely to remain within the muscle cone and can be made sufficiently large to maintain satisfactory orientation of orbital structures, rather than because of any particular shape imparted to the stump. Long experience with spherical implants has shown that tissue between the implant and prosthesis will gradually erode through, unless pressure is distributed over a considerable area and the intervening tissue is of reasonable thickness. None of the newer "motility" implants have been used long enough for true evaluation of permanency. (15 references)

Hughes, Wendell L. **Classification and mechanics involved.** pp. 25-27.

The author uses two main types of implants to be placed into the orbit, buried implants and open-face implants, in which an anterior part of the implant is intentionally left uncovered. He describes the mechanics involved in enucleation and evisceration using the various types of implants.

Cutler, N. L. **Principles underlying the surgical technic.** pp. 28-29.

The first principle to consider for any type of implant is the proper selection of the patient. Unfavorable types of patients are 1. elderly individuals, and deep set eyes with narrow fissures; 2. individuals with contracted or scarred sockets; 3. patients who live or work in a dusty atmosphere; 4. patients who cannot be properly followed; 5. patients who will not follow a good hygiene (primarily in reference to integrated implants); 6. patients with a tumor that may extend outside the eye. Favorable for implants are younger individuals; normal or near-normal sockets; and patients who can be seen by an ophthalmologist at intervals and who will maintain proper hygiene of eye and socket. Basic principles of implant surgery in the motility type of implant are muscle dissection and muscle fixation and smooth application of the conjunctival edge around the neck of the implant. The conjunctival covering should be preserved.

Troutman, Richard C. **End results of implant surgery.** pp. 30-34.

The author presents twelve tables, the result of a brief statistical report compiled on material gathered through a survey on implants sponsored by the Academy.

Stone, William, Jr. **Causes of complications and their solution.** pp. 35-41.

The five main complications are due to: 1. secretion; 2. uncovering in the case of the buried implant or exposure of meshwork or plastic in the case of the incompletely covered implant; 3. granulation tissue; 4. operative difficulties with secondary or delayed implants; and 5. difficulties with fitting shells to allow full motility. If incompletely covered implants are made correctly, if cleanliness of the socket is maintained, and if operative technique is correct, the results with the integrated implants of the present variety should be satisfactory. With buried implants, if tension of the conjunctiva is relieved, if cleanliness of the socket is main-

tained, and if no undue pressure is placed on the conjunctiva, results should be satisfactory. In the aged, indigent people who cannot maintain proper cleanliness of their sockets, incompletely covered implant should not be used. Some form of buried implants should be used for these cases. (2 tables, 5 figures)

Theodore M. Shapira.

Gorduren, S. **Orbital encephalocele associated with acrocephaly.** Brit. J. Ophth. **36**:151-154, March, 1952.

Orbital encephalocele is a congenital tumor which remains in communication with the intracranial cavity and is probably due to an anomaly of ossification of the skull. Other anomalies of the skull are usually coexistant. An orbital encephalocele is described which actually had a branch in each orbit and pushed each eye laterally. The diagnosis was confirmed by needle-puncture aspiration and by X-ray studies. No method of treatment is given. (6 figures) Morris Kaplan.

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TUMORS

Marconcini, Eraldo. **Basal-cell carcinoma of the eyelid probably of traumatic origin.** Arch. di ottal. **55**:623-631, Nov.-Dec., 1951.

A patient, 81 years of age, had been wounded in the lower lid three years previously by a shell fragment. A tumor developed at the site of the injury and was removed by plastic surgery. It proved to be a basal-cell carcinoma. The literature is discussed. The trauma was the probable cause of the development of the carcinoma. John J. Stern.

Stankovitch, M. **Sarcoma of the four lids.** Arch. d'opht. **12**:58-60, 1952.

A woman, 60 years of age, had tumefaction of all four lids of two and one-half months duration. Both skin and conjunctiva were adherent to the tumors, which had a waxy consistency. There was severe preauricular and submaxillary adenopathy; the globes showed no abnormalities except dilation of the retinal veins. The blood was normal. Biopsy showed a small round-cell sarcoma. Death occurred six months later; the submaxillary glands had by this time reached enormous proportions. Involvement of all four lids in this type of tumor is rare.

P. Thygeson.

18

SYSTEMIC DISEASE AND PARASITES

Mariotti, Lorenzo. **Ocular symptoms in tuberculous meningitis** (clinical and statistical considerations with regard to antibiotic therapy). Ann. di ottal. e clin. ocul. **78**:25-32, Jan., 1952.

Ocular signs were observed in over 400 cases of tuberculous meningitis treated with streptomycin and para-aminosalicyclic acid. There was hyperesthesia in 20 percent of the patients, photophobia in 40 percent, disturbances of intraocular and extraocular motility in 30 and 45 percent respectively. Lesions of the optic nerve occurred in 72 percent and in the retina and choroid in 29 percent. Regular, systematic examination of the eyes is essential in the proper mangement of tuberculous meningitis. (1 table, 16 references)

Harry K. Messenger.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

DEATHS

Dr. Harold Bailey, Charles City, Iowa, died March 9, 1952, aged 77 years.

Dr. Ralph Edwin Boice, Toledo, Ohio, died March 30, 1952, aged 56 years.

Dr. Robert Kahn, New York City, died March 26, 1952, aged 67 years.

Dr. William Sassaman Sutherland, Philadelphia, died March 14, 1952, aged 55 years.

ANNOUNCEMENTS

DIRECTORY AVAILABLE

The American Foundation for the Blind announces the publication of the ninth edition of the *Directory of Activities for the Blind in the United States and Canada*, compiled by Helga Lende, the Foundation's librarian. The Directory, which first appeared in 1932, is published biennially. National agencies, public and private, are listed first, followed by state and local agencies, listed by states. The price is $2.00, postpaid. The accompanying chart and list showing geographical areas served by the 28 distributing libraries for the Library of Congress in lending Braille and talking books is most useful.

The Directory always has been looked upon as an invaluable reference book for all workers for the blind, as well as for those in the field of public welfare. It provides information for individuals who seek to learn where they may turn for assistance for blind friends or relatives. It gives correct names, addresses, and executives of every important agency for the blind in the United States and Canada with other pertinent information regarding these agencies.

RESEARCH FELLOWSHIPS

Information regarding research fellowships in ophthalmology at Indiana University Medical Center may be obtained by writing to Dr. T. F. Schlaegel, Jr., 1040 West Michigan Street, Indianapolis 7, Indiana. It is not necessary to have had training in ophthalmology to be accepted for these positions. For those who have not had a graduate course in ophthalmology, these research fellowships will provide some training prior to a residency. The fellows assist in the eye clinic and at eye surgery and attend the formal classes for eye residents. The stipend is $150.00 per month plus laundry.

REFRESHER COURSE IN EYE SURGERY

The Faculty of Medicine of the University of Toronto offers a refresher course in eye surgery from April 13 to 17, 1953. The instruction will consist of lectures, operative clinics on patients and cadaver surgery in small groups.

The guest surgeons will be: Dr. Dohrmann K. Pischell, San Francisco, and Mr. H. B. Stallard, F.R.C.S., London, England.

The staff of the Department of Ophthalmology in the University will contribute extensively to the course. The course will be given for a minimum of 10 and a maximum of 30 students. The fee will be $100.00 payable to the chief accountant, Simcoe Hall, University of Toronto. Applications should be made to the Dean of the Faculty of Medicine, not later than January 31, 1953.

EGYPTIAN SOCIETY OFFERS PRIZE

The Ophthalmological Society of Egypt offers a prize for the most valuable contribution brought before the annual congress of the society. For further information write: Dar El Hekma, 42 Kasr El Ainy Street, Cairo.

GRADUATE TRAINING

The University of Toronto, Faculty of Medicine offers a postgraduate course in ophthalmology extending over three years. The graduate instruction in ophthalmology in the teaching hospitals in Toronto has been coördinated under the direction of the university. The first year on a fellowship, the value of which is approximately $1,400.00, the student spends in one of the basic sciences of ophthalmology, and the final two years are spent on the intern service of one or more of the university teaching hospitals. Approximatey four hours of didactic teaching are arranged for the students by members of the staff each week from October to May. Teaching ward rounds are made at the Toronto General Hospital and are attended by the interns from the other university hospitals.

A sound knowledge of neurology and metabolic diseases is desirable. The following courses are given: geometric and physiological optics, physiology of the eye, principles and practice of biomicroscopy, perimetry, ocular therapeutics, medical ophthalmology, pathology of the eye, bacteriology and external diseases of the eye, embryology and developmental anomalies of the eye, ocular motor anomalies, anatomy of the orbit, skull and brain, radiological ophthalmology, industrial ophthalmology, intraocular and plastic surgery, pathology of the visual pathways, refraction, neuro-ophthalmology, glaucoma. Senior interns are given instruction in the preparation and presentation of scientific papers.

The fee for instruction is $50.00 per year payable to the chief accountant, University of Toronto.

An application for appointment may be made to the professor of ophthalmology, Faculty of Medicine, University of Toronto. Appointments are made in December to commence on the following July 1st.

Miscellaneous

Teaching materials needed

In collaboration with the Gandhi Eye Hospital, an Institute of Ophthalmology has been established at Muslim University, Aligarh, U.P., India. The institute, which is for graduate study, is directed by Prof. Alfred Leber, formerly of the University of Goettingen.

The institute would greatly appreciate receiving material from America, especially slides. Text books, charts, laboratory equipment, and so forth, are very difficult to get in India, and surplus texts and other material would be useful, even if not the latest editions. The material can be sent direct to Dr. Zakir Husain, vice chancellor, Muslim University, Aligarh, U.P., India, or if more convenient it may be sent to Arthur E. Morgan, P.O. Box 243, Yellow Springs, Ohio.

Muslim University in Aligarh is the only Moslem university in India, and it receives students from most parts of the Moslem world.

Richmond Eye Hospital

On May 12th, the Richmond (Virginia) Eye Hospital was officially opened. Although this hospital was named the Richmond Eye Hospital because of certain desires of the original contributor to the institution, approval was obtained from the executors to include among its patients those generally classified in the branches of otolaryngology.

Mrs. Virginia Fox Beveridge of Richmond, who first became interested in building an eye hospital, bequeathed a substantial sum of money "for the establishment and the endowment of an eye hospital in the city of Richmond." She directed the executors of her will to carry out her wishes and named the first board of directors to serve with the executors.

Shortly after the bequest of Mrs. Beveridge was revealed, two anonymous benefactors contributed large sums which, with numerous small donations from other individuals and firms, made possible definite ideas of construction. With the coöperation of the authorities of the Medical College of Virginia, a plot of ground, situated directly across a street from the teaching hospitals and out-patient clinics of the Medical College of Virginia and connected with these institutions by an underground passageway, was acquired by the Eye Foundation. Although the Richmond Eye Hospital will receive some services from the Medical College of Virginia Hospital, it will operate under a separate board of directors. At the same time, its staff will contribute to the training of postgraduate students of the main teaching hospital.

At the present time the building has three floors above the ground level with an added penthouse,

Richmond Eye Hospital

a basement, and sub-basement. On the first floor are the administrative offices, four operating rooms, nurses supply and sterilizing rooms. The second and third floors which are for patients have from 20 to 26 beds. In the penthouse are rooms for the residents and records rooms. The basement contains a modern kitchen, separate dining rooms for the nurses and personnel and the air-conditioning units. The sub-basement is occupied by the laboratory, the heating, X-ray laboratories, and other such equipment.

Societies

Illinois meeting

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Abraham Lincoln Hotel in Springfield, Illinois, on September 12th, 13th, and 14th. The guest speakers will be Dr. Paul A. Chandler of Boston, Massachusetts, and Dr. Thomas C. Galloway of Evanston, Illinois.

Colorado officers

At a recent meeting of the Colorado Ophthalmological Society in Denver, the following officers were elected for the coming year: President, Dr. Fritz Nelson, Colorado Springs; vice-president, Dr. Samuel Goldhammer, Denver; secretary, Dr. James Strong, Denver; treasurer, Dr. George Filmer, Denver; recorder, Dr. Daniel Franklin, Denver.

St. Louis officers

At the annual business meeting of the St. Louis Ophthalmic Society, the following officers were elected for the year 1952-53:

President, Dr. Edmund B. Alvis; vice-president, Dr. Robert D. Mattis; secretary, Dr. Benjamin Milder; treasurer, Dr. A. G. Boldizar; editor, Dr. Edwin Casey.

Oxford program

At the 37th annual meeting of the Oxford Ophthalmological Congress held on July 3rd, 4th, and 5th, the following program was presented:

Mr. F. A. Williamson-Noble, London, "Venous pulsation"; Dr. Bernard Samuels, New York, "Internal gaping of corneal wounds after injuries and operations"; Prof. G. B. Bietti, Parma, Italy, "The results of audiometric examinations in some ocular diseases"; Mr. J. H. Doggart, London, "Crystals in the posterior segment of the eye." Dr. Raul Arganaraz, Buenos Aires, presented films on "Corneal graft operation" and "Modern operation of senile cataract."

Mr. G. T. Willoughby Cashell, Reading, and Mr. A. A. Douglas, Perth, were openers for a discussion on "Long term results of treatment of concomitant convergent strabismus in terms of binocular function," and Mr. T. H. Whittington, London, Mr. A. C. L. Houlton, Oxford, and Mr. Keith Lyle, London, took part in the discussion. Mr. H. B. Stallard, London, showed a film on "The use of radium discs in the treatment of malignant intraocular neoplasms."

Dr. D. Harley, London, discussed "Allergic aspects of iridocyclitis"; Mr. O. Gayer Morgan, London, "Methyl alcohol addiction"; Mr. A. McCurry, Leicester, "An ophthalmologist and a fever hospital"; Mr. Harold Ridley, London, "Some recent methods of fundus examination" and "A further report on intraocular acrylic lenses"; Prof. A. Franceschetti, Geneva, Switzerland, "Electronystagmography by analysis of congenital nystagmus."

Mr. E. B. Alabaster, Birmingham, "Saccharopathies"; Dr. Thomson Henderson, Nottingham, "How comes the physiology of the choroid, ciliary muscle and intraocular pressure to be unlike that elswhere?"

For the discussion on "Recent trends in ocular therapeutics," Mr. Frederick Ridley, London, Prof. E. J. Wayne, Sheffield, and Mr. A. Stanworth, Manchester, acted as openers. Following a film entitled "A new form of plastic implant" shown by Dr. Stanislaw Gebertt, Coventry, a discussion on "Plastic implants," was lead by Mr. J. Horton Young, Nottingham.

The Doyne Memorial Lecture was delivered by Dr. Dorothy Campbell, Coventry, whose subject was "Ophthalmic stress."

Personals

At the annual meeting of the University of Colorado Medical Alumni Association, Dr. Ralph W. Danielson, Denver, head of the division of ophthalmology, was one of five professors given 25-year service awards.

Dr. Frederick H. Verhoeff, Boston, has been made a colonel on the staff of the Governor of Kentucky.

Dr. William E. Krewson, 3rd, Philadelphia, has been appointed director of the departments of ocular motility and perimetry at the Wills Eye Hospital.

Dr. Arlington C. Krause, associate professor of ophthalmology, University of Chicago Medical School, has been elected president of the Chicago Orthoptic Society.

Dr. Alston Callahan, professor of ophthalmology at the Medical College of Alabama, Birmingham, recently visited South America, his tour being sponsored by the Pan-American Association of Ophthalmology. Dr. Callahan visited Lima and then flew to São Paulo, where he gave two lectures at the Centro de Estudos de Ophtalmologia. His subjects were "Reparative surgery of the eyelids" and "Management of penetrating and blunt injuries of the eye." He then went on to Rio, from Rio to Caracas, Venezuela, and thence back to the United States.

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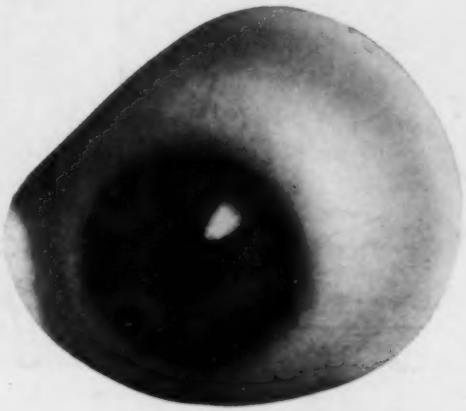

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